EXPERIMENTAL STUDIES ON THE EFFECTS OF EXERCISE ON THE LACTATE AND VENTILATORY THRESHOLD

by

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PREFACE

In order for an athlete to perform exercise for a prolonged period of time, the cardiovascular, respiratory and muscular systems must function together in an efficient manner. The most widely used method to measure these endurance parameters has been maximal oxygen consumption (VO_2 max) (Astrand and Rodahl, 1977). An individuals ability to consume and utilize large quantities of O_2 reflect an efficient system. Research suggests that as the exercise duration increases, aerobic capacity becomes increasingly more important (MacDougall, 1977).

Recent research however, has also demonstrated that the 'anaerobic threshold' (AT) is an important aerobic parameter (Sjodin et al., 1982). The ability of an athlete to sustain exercise at a higher percentage of VO_2 max, without accumulating metabolites, may be as important as VO_2 max values (Thoden et al., 1983; Weltman et al., 1978). The concept of AT was originally used to describe the metabolic events associated with the production of lactic acid during progressive exercise. However, in this attempt researchers have encountered a number of problems associated with the anaerobic threshold. These include: an appropriate definition or terminology (Kindermann et al., 1979; Skinner and McLellan, 1980), validation and determination of a threshold measurement (Yeh et al., 1983), and problems associated with its use (Jones and Ehrsam, 1982).

The anaerobic threshold has been defined as the intensity of exercise where blood lactate begins to accumulate in blood (MacDougall, 1977). It has been suggested that this metabolic phenomenon is due to an imbalance between pyruvate production and utilization in the Krebs cycle (Holloszy, 1975). Pyruvate is converted to lactate in the presence of lactate dehydrogenase (LDH) and reduced nicotinamide adenine dinucleotide (NADH), in an attempt to regenerate NAD^+ to maintain production of ATP via glycolysis. During lactate production, hydrogen ions (H^+) are formed in an equimolar concentration, creating a metabolic acidosis which then must be buffered. The bicarbonate buffering system in blood controls pH levels when H^+ and HCO_3^- combine, and the excess CO_2 produced (Hughson and Green, 1982) then stimulates ventilation (V_E). As exercise intensity increases, an equivalent fall in bicarbonate concentration continues as lactate levels rise (Jones, 1980). This results in further hypernea due to the stimulation of respiratory chemoreceptors by increases in P_{CO2} and H^+. Therefore, it has been suggested that noninvasive gas measures can be used to reflect lactate and H^+ formation, and consequently metabolic acidosis (Davis et al., 1976; Wasserman et al., 1973). Thus, a ventilation threshold (V_E vs VO_2 or V_E/VO_2 vs VO_2) has been used to reflect the lactate threshold (LA vs VO_2). It is this event which can then limit endurance performance.

Recent research however, suggests that it is unreliable to use a respiratory event to reflect a metabolic phenomenon (Green et al., 1983). These authors conclude that this relationship between ventilation and metabolism was only coincidental. Some researchers have explored the physiological bases of the anaerobic threshold in an attempt to determine what factors effect the lactate (LT) and ventilation (VT) thresholds. Hughes et al. (1982) studied the effects of glycogen depletion exercise on LT and VT and found these thresholds could be manipulated independently of each other. This suggests that the relationship is not one of cause and effect. Stamford et al. (1978a) discussed the anaerobic threshold and cardiovascular responses during one- versus two-legged cycling and suggested that AT was similar between leg protocols. They did not however, discuss to what extent peripheral and central (circulatory) factors play on the exercise responses at the LT and VT. This may help to shed light on the nature of the training stimulus. There is limited research on the effects of different protocols on the measurement of LT and VT (McLellan, 1983). Documenting these effects may provide greater insights into the nature of lactate efflux from muscle during resting periods when using an intermittent protocol.

With this background information, the purpose of the present studies were:

1. to examine the effects of a continuous and discontinuous exercise protocol on the lactate and ventilation thresholds.

2. to determine the differences between 1- versus 2-legged cycling on the lactate and ventilation thresholds and on selected variables at VO_2 max.

3. to determine both the reliability of the lactate and ventilation thresholds and the effects of prior exercise on these threshold points.

4. to determine whether the relationship between the lactate and ventilation threshold is coincidental or cause and effect.

We accept this thesis as conforming
to the required standard

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CHAPTER I

THE EFFECTS OF CONTINUOUS VERSUS DISCONTINUOUS CYCLING ON THE LACTATE AND VENTILATORY THRESHOLD

Abstract

The lactate (LT) and ventilation threshold (VT) were examined in three male university students under a continuous (C) and discontinuous (D) exercise test protocol on a cycle ergometer. The LT was determined invasively from the 'breakaway' increase in venous lactate (LA) above resting levels, while the VT was determined noninvasively from the non-linear increase in ventilation (V_E). Power output (PO) was increased 30 W every 3 minutes. Venous blood samples were drawn during the last 30 s of each load. Expired gases were measured every 30 s (Beckman Metabolic Measurement Cart). LT was found to be significantly different ($p<0.05$) between the C and D protocols (4.25 + 0.43 mmol l^{-1} and 2.93 + 0.18 mmol l^{-1}, respectively). VT showed no significant differences between test protocols (C = 2.82 l min^{-1}, D = 2.89 l min^{-1}). In addition, LT and VT were shown to occur at the same time for both protocols. These results suggest that the exercise protocols do affect the measurement of LT but not VT.

Introduction

The type of exercise protocol employed in laboratory research is generally dependent upon the purpose of the study (Cardus, 1979). The exercise test can be either continuous or discontinuous, with uni- or multi-stepped loads, ramped or sinusoidal increases (Cardus, 1979). Research has shown that the test protocol used will effect the outcome of the physiological variables (i.e., VO_2 max, VCO_2, HR, VT and LT) (Hughson and Green, 1982; Jacobs et al., 1983; Skinner and McLellan, 1980; McLellan, 1983; Hagberg et al., 1981; Hughes et al., 1982). Thus, no single protocol has been deemed appropriate for all types of testing and/or subjects and there has been little concensus on the selection of continuous versus discontinuous loads regardless of the exercise mode employed. It has been suggested that the discontinuous protocol be used primarily when testing untrained subjects, while continuous be employed for experienced and trained subjects (Cardus, 1979).

Therefore, this study was undertaken to determine whether a continuous or discontinuous exercise protocol will affect the venous lactate threshold and ventilatory threshold in active healthy subjects.

Methods

Three healthy volunteer male physical education students signed informed consent and took part in this study. Their physical characteristics are shown in Table 1. All subjects were familiar with the laboratory procedures and the physical demand required.

The subjects performed two separate submaximal (85 - 90% VO_2 max) tests to determine lactate and ventilation thresholds. A calibrated stationary Monark cycle ergometer was used for all exercise testing. The first test was a continuous progressive exercise ride consisting of 3 minute loads (MacDougall et al., 1983). Each subject began cycling at 60 watts (W) during the warm-up. Thereafter, power output (PO) was increased 30 W every 3 minutes until 180 W. Work increments were then increased 16 W until termination of the test. Subjects maintained a pedalling frequency of approximately 60 rpm with revolutions counted mechanically. The second test, a discontinuous protocol, was performed on a separate day and included the same 3 minute work duration and PO increments but 3 minute rest periods were interspersed (1:1 work-rest ratio).

During the exercise tests, subjects breathed through a low-resistance respiratory valve and expired O_2 and CO_2 were collected and analyzed using the Beckman Metabolic Measurement Cart (MMC). The gas analyzers were calibrated before each test with gases of a known concentration. Physiological measures, including V_E, VCO_2, VO_2, R and HR were recorded every 30 s.

Prior to each test a 20 gauge Teflon catheter (Jelco Cathlon, 3.2 cm) was inserted into a superficial arm vein (median vein or medial cephalic) and taped in place for periodic blood sampling. Venous blood samples were taken at rest, immediately following the warm-up, and during the last 30 s of each load throughout exercise. The blood samples (0.50 ml) were immediately deproteinized in ice-cold perchloric acid (2.0 ml) for subsequent analysis of lactate concentrations employing the spectrophotometric technique (Sigma Chemical Company, 1981).

The criterion for determination of LT and VT was a non-linear increase in venous lactate concentration and V_E vs VO_2, respectively (MacDougall et al., 1983).

A 1-tailed students t-test was employed to determine significant differences ($p < 0.05$) between threshold values.

Results

The power output and VO_2 ($l\ min^{-1}$) at LT and VT during the C and D exercise protocols are presented in Table 2. The exponential rise in venous lactate concentration (mmol l^{-1}) occurred at 4.25 + 0.43 and 2.93 + 0.18 mmol l^{-1} ($p < 0.05$) during the C and D protocols, respectively.

During the continuous cycle ergometer ride, the LT and VT occurred at 2.95 and 2.82 l min^{-1} respectively, whereas, during the discontinuous protocol they occurred at 2.84 and 2.89 l min^{-1} (Fig 1).

The submaximal cycle ergometer rides illustrated a different response in venous LA concentration amongst the three subjects. At LT, venous LA concentrations varied from 4.0 to 4.50 mmol l^{-1} for the continuous protocol, and from 2.59 to 3.24 mmol l^{-1} for the discontinuous protocol.

No significant difference in HR was observed between the two different protocols.

Discussion

These data indicate that significant differences exist between the continuous and discontinuous test protocols for venous LA threshold, but no significant differences in ventilatory measures between the two protocols.

The finding that ventilatory measures during the continuous and discontinuous test protocols were not significantly different are in agreement with Gleser and Vogel (1971) and McArdle et al. (1973). Results also indicate that venous LA concentration increased with the onset of exercise during both C and D cycling. It was found that the lactate concentration at low intensity exercise (< 65% VO_2 max) was similar. The LA concentration at LT, however, was different ($p < 0.05$) between protocols (Fig 1).

During low power outputs the aerobic system is capable of supplying sufficient energy to maintain a steady state, and thus LA formation is minimal. When exercise intensity is increased to 60-70% VO_2 max, more energy is supplied anaerobically leading to the appearance of elevated lactate levels in blood (Kindermann et al., 1979). Lactate at this point is approximately 2 - 4 mmol l^{-1} (Skinner and McLellan, 1980). The increased R value during this phase of exercise reflects this phenomenon. However, during the D test a lower R value was observed at the higher loads (80-85% VO_2 max), suggesting a lower level of anaerobic metabolism in comparison to the C test (McArdle et al., 1973).

Research indicates that during the rest periods in interval work, free fatty acid (FFA) oxidation is increased and remains elevated at the end of each rest period (Essen, 1977). Parmeggiani and Bowman (1963) showed that citrate concentration remained elevated following each rest period during intermittent exercise suggesting that high citrate concentrations may inhibit PFK enzyme in glycolysis, and therefore, increase lipid oxidation (Essen, 1977). Animal studies have also demonstrated that FFA utilization is occurring during intermittent exercise even at high work intensities (Snow et al., 1983). Therefore, the use of triglycerides as an energy source creates a glycogen sparing-effect within the muscles and thereby reducing the formation of lactate (Gollnick, 1977). Also, ATP, CP and oxymyoglobin stores are replenished during these periods of rest making them available as an energy source for the next work bout. Therefore, the combination of these processes reduce lactate formation during intermittent exercise.

The threshold level occurred at approximately the same VO_2 for both venous lactate and ventilation. This is in agreement with Davis et al. (1976); Wasserman et al. (1973); and Ivy et al. (1980) who concluded that the point at which blood LA begins to accumulate corresponds closely to the point at which V_E demonstrates an abrupt increase from linearity. Studies by Taylor and Jones (1979), have demonstrated that there appears to be a linear relationship between plasma LA concentration and CO_2 output. This relationship exists because LA accumulation in plasma is accompanied by hydrogen ion (H^+) efflux from the muscle. The H^+ efflux increases CO_2 output which stimulates an increase in V_E (Whipp, 1978). This association allows us to use the ventilation threshold as an indication of the lactate threshold (Jones and Ehrsam, 1982). Since LT and VT occur at the same VO_2 in both protocols but venous LA is lower in the discontinuous protocol, it suggests that factors other than venous lactate and its associated changes in P_{CO2} and pH may also affect the VT. Others have also discussed problems with the validity of VT (Davis et al., 1976; Green et al., 1983; Yeh et al., 1983).

This study suggests that there is no difference in VO_2 (l min^{-1}) at the LT and VT, and no difference between the VT in a continuous or discontinuous protocol. However, the venous LA concentration at LT is lower with the discontinuous protocol. Therefore, although the power output and VO_2 at both thresholds is not affected by either protocol, if venous LA concentration is the sole criterion, then the type of protocol selected must be carefully considered.

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Table 1. Physical characteristics of the subjects.

Subject	Age (year)	Weight (kg)	Height (cm)	VO_2 max ($l\ min^{-1}$)	VO_2 max ($ml\ kg^{-1}\ min^{-1}$)
1	20	69.3	175	4.45	64.2
2	26	65.4	173	3.90	59.6
3	23	75.8	171	3.87	51.1

Table 2. Venous lactate (LA), oxygen consumption (VO_2), power output (PO), and heart rate (HR) at both lactate and ventilation thresholds for each protocol.

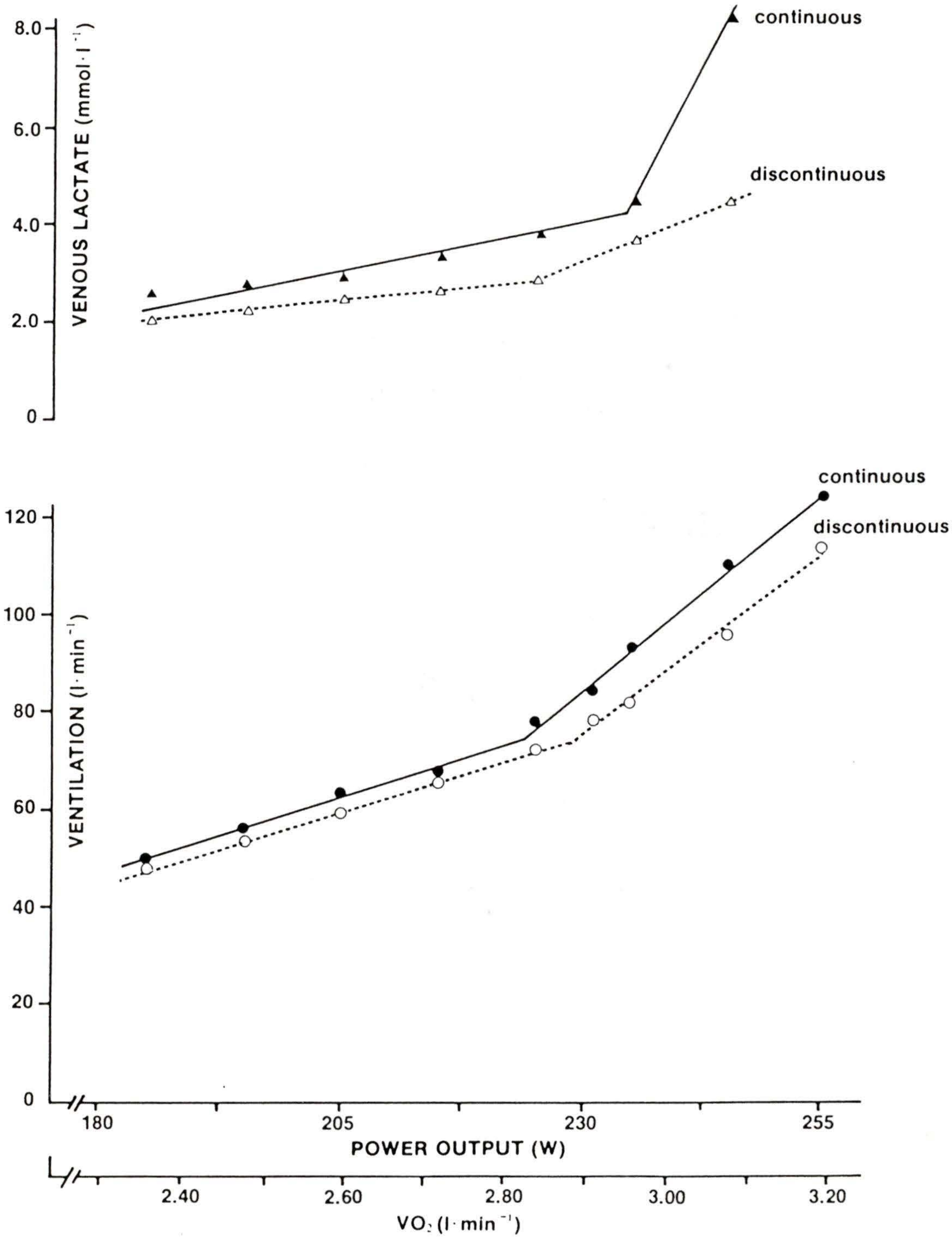
	LA (mmol l^{-1})	VO_2 (l min^{-1})		PO (Watts)		HR (bpm)
Protocol	LT	LT	VT	LT	VT	VT
C	4.25^*	2.95	2.82	235	220	174.0
	0.43	0.20	0.18	7	2	2.9
D	2.93^*	2.84	2.89	224	230	165.0
	0.18	0.15	0.16	6	3	7.6

Values are means + SE

* significance at $p < 0.05$

Figure 1 - Mean lactate and ventilation threshold for the continuous and discontinuous protocols

Figure 1:
Mean lactate and ventilation threshold for the continuous and discontinuous protocols.



CHAPTER II

THE EFFECTS OF ONE- AND TWO-LEGGED EXERCISE ON THE LACTATE AND VENTILATORY THRESHOLD

Abstract

The purpose of this investigation was to compare differences between one- and two-legged exercise on the lactate (LT) and ventilation (VT) threshold. On four separate occasions, ten male volunteer subjects (1-leg VO_2 max = 3.36 l min^{-1}; 2-leg VO_2 max = 4.27 l min^{-1}) cycled to determine 1- and 2-legged submaximal and maximal measurements. The submaximal threshold tests for 1- and 2-legs, began an with a warm-up at 50 W and then increased every 3 min by 16 W and 50 W, respectively. Similar increments occurred every minute for the maximal tests. Venous blood samples were collected during the last 30 s of each work load, whereas noninvasive gas measures were calculated every 30 s (Beckman Metabolic Measurement Cart). No differences in VO_2 (l min^{-1}) were found between 1- and 2-legs at LT or VT, but significant differences ($p < 0.05$) were recorded at any power output. Lactate (LA) concentration was different between 1- and 2-legged threshold tests (2.62 mmol l^{-1} vs. 1.97 mmol l^{-1}) at LT. Heart rate (HR) and R were similar between legs at LT and VT, however, significant differences occurred at power outputs greater than 50 W between leg protocols. Maximal 1-legged performance measures for VO_2, V_E and HR reached 79%, 86% and 92% of their corresponding 2-legged measurements, respectively. These results suggest it is VO_2 or oxygen cost rather than muscle mass which affect LT and VT, and that the central circulation is limiting to VO_2 max.

Introduction

Previous research has employed one- and two-legged models (Davies & Sargeant, 1974; Gleser, 1973), trained and untrained limbs (Saltin et al., 1976), arm versus leg exercise (Clausen et al., 1973) and a combination of these models (Stamford et al., 1978b; Clausen et al., 1976), to demonstrate whether aerobic training effects reside primarily in the central circulation or in the peripheral muscle. Although it has been suggested that cardiovascular limitations imposed upon two-legged exercise do not apply to maximal one-legged exercise (Stamford et al., 1978b), a general concensus has not been reached as to what extent central and peripheral components effect and limit maximal work.

Research has also studied the physiological response to submaximal exercise during one- and two-legged cycling. Stamford et al. (1978a), demonstrated that the 'anaerobic threshold' (AT) occurred at the same VO_2 between 1- and 2-legs, when expressed as a % VO_2 max. These authors concluded that the size of the exercising muscle mass was not related to 'breakaway' but possibly to fiber type, aerobic and anaerobic muscle potential, or local factors. However, in a study by Davis et al. (1976) using three different modes of exercise, AT occurred at 59%, 64% and 47% VO_2 max for treadmill running, leg cycling and arm cranking, respectively. They concluded that differences in threshold values were due primarily to the size of the muscle mass or unfamiliarity of exercise performed. Further research to resolve this conflict is needed.

Therefore, the purposes of this study were to investigate the effects of one- and two-legged exercise on the lactate (LT) and ventilation (VT) threshold; and on selected physiological responses during maximal work.

Methods

Subjects

Ten healthy male subjects (mean 2-legged VO_2 max = 4.27 l min^{-1}) volunteered to perform 1- and 2-legged stationary cycling. Informed consent was obtained from the subjects, following familiarization with the equipment, testing procedures, and one-legged cycling. The physical characteristics of the subjects are presented in Table 1. Seven of the ten subjects were members of different university varsity teams.

Procedures

The testing schedule is presented in Fig 1. All testing was completed on 4 separate days within 3 weeks.

The VO_2 max tests performed on a Monarch cycle ergometer, consisted of a two minute warmup at 50 W, after which time the load was increased 16 W and 50 W every minute thereafter for one- and two-legs, respectively. The criteria for VO_2 max included: less than a 100 ml increase in VO_2; a levelling off and then a decrease in VO_2; or voluntary termination of the test. Both tests lasted 10-15 minutes.

The 1- and 2-legged LT and VT tests involved a 3 min. continuous protocol on an electrically braked ergometer (Quinton Instruments, model 844). Threshold tests included a warm-up at 50 W and then 16 W increments thereafter. The pedalling cadence was maintained at 60-70 revolutions per min (rpm) throughout the 15-24 minute test. All subjects were requested to refrain from their regular physical activity the day prior to each test. Subjects breathed through a low resistance respiratory valve, during which time respiratory and metabolic measures (V_E, VO_2, VCO_2, FE_{CO2}, FE_{O2}, R) were monitored every 30 s (Beckman Metabolic Measurement Cart). The gas analyzers were calibrated prior to each test with known gases. Heart rate (HR) was monitored each minute.

During one-legged cycling, a toe clip and heel strap were used to secure the foot. The non-working leg was rested on the cycle ergometer between the pedals.

Venous blood samples were taken via an indwelling catheter (Angio-Cath, 20g x 3.2 cm) placed in the antecubital vein. Blood samples (0.50 ml) were quickly deproteinized in 2.0 ml of ice cold 4% perchloric acid, and later analyzed spectrophotometrically for lactate concentration (Sigma Lactate Kit; Sigma Chemical Company, 1981).

The criteria for determination of lactate and ventilation thresholds was a non-linear increase in LA vs VO_2, and V_E vs VO_2, respectively. Individual LT and VT breakaway points were determined using a linear regression model, and then group means calculated. The data was treated using a pairwise Student's t-tests. The alpha level for significance was set at $p < 0.05$.

Results

The metabolic and respiratory responses to one- and two-legged cycling at LT and VT are found in Table 2, and differences between work loads are presented in Table 3.

During exercise, VO_2 (l min^{-1}) responses were the same ($p > 0.05$) between 1- and 2-legged cycling at LT and VT (Table 2). VO_2 (l min^{-1}) however, was significantly greater at each power output (PO) for 1-legged exercise (Table 3; Fig 2). Ventilation (V_E) was also higher ($p < 0.05$) at any given power output for 1- compared to 2-legged work, but was not different at VT (Fig 3).

Venous lactate concentration demonstrated a characteristic curvilinear increase with increasing VO_2 (l min^{-1}). When LA concentrations were compared at a given PO, values were significantly higher for 1-leg ($p < 0.05$) at 100 W and 150 W (Fig 4). No differences existed at rest or at 50 W. In addition to higher lactate values, R was also higher ($p < 0.05$) at 100 W and 150 W. VCO_2 (l min^{-1}) therefore increased with increasing PO. R, however, was the same at threshold values for 1-leg and 2-leg cycling.

Heart rate responses to exercise demonstrated a linear relationship to increasing VO_2. At LT and VT no differences were observed between leg protocols, but was higher ($p < 0.05$) for 1-leg at 100 W and 150 W (Fig 5).

Maximal performance: During 1-legged maximal cycling, VO_2, V_E and HR were 79%, 86%, and 92% of their respective 2-legged maximal values ($p < 0.05$) (Table 4).

Discussion

The present study was undertaken to determine if differences in selected physiological variables exist between 1- and 2-legged cycling at LT, VT, and at VO_2 max.

Oxygen consumption and ventilation (l min^{-1}) were found to be significantly higher ($p < 0.05$) at any given power output during 1-legged exercise which confirms results obtained by Stamford et al. (1978a) and Freyschuss and Strandell (1968). Since one-legged cycling required a greater VO_2 at all comparable power outputs, it is assumed that a reduced mechanical efficiency represents the discrepancy between 1- and 2-legged exercise, as indicated by the relationship between VO_2 and PO (Davies and Sargeant, 1974). During 2-legged cycling, both legs are engaged to help with the upward and downward stroke; but during 1-legged exercise the work must be performed only by the single limb. Therefore, the contracting muscles are required to apply more force throughout a greater range. Sargeant and Davies (1977) have also suggested that a postural component may contribute to metabolic differences between legs.

In regards to VO_2, our results show some discrepancy with Stamford et al. (1978b). At 50 W, exercise intensity represented approximately 32% VO_2 max and 19% VO_2 max for 1- and 2-legs, respectively. They demonstrated values of 47% and 36% VO_2 max, respectively. This difference was probably due to the fitness level of the subjects. The difference in protocol used in these studies would also account for changes at the specific power outputs. Unlike other 1-legged studies, a toe clip and heel strap were used in this laboratory. It has been reported that higher maximal power measures are recorded with this addition (LaVoie et al., 1984).

At LT and VT, VO_2 and V_E ($l\ min^{-1}$) were similar between single and double-legged exercise. Previous work in this laboratory (Neary and Wenger, unpublished data) has also demonstrated that VO_2 ($l\ min^{-1}$) was similar at both thresholds even at different induced blood lactate levels. This suggests that the breakaway in VE and blood lactate may be due to shifts in the neural recruitment of muscle for a specific O_2 cost or power output.

Stamford et al. (1978a) and Davis et al. (1976), and the present data demonstrate that the VO_2 at given power outputs is higher for 1-legged effort. Thus the efficiency of 1-legged exercise on the cycle ergometer is lower than in 2-legged work. However, since the VO_2 at the thresholds was similar, this suggests that the VO_2 or oxygen cost is related to the triggering of the LT and VT.

Blood lactate accumulation in any appreciable amount, generally does not occur at work intensities less than 50% VO_2 max (Stamford et al., 1978c). This however, was not the case during 1-legged exercise. Results showed that LA concentration was higher ($p < 0.05$) at 100 W (50% VO_2 max) and above. At 100 W, LA concentrations were 2.40 mmol l^{-1} and 1.68 mmol l^{-1}, respectively, for 1- and 2-legs. These results are consistent with Stamford et al. (1978a) and Davies and Sargeant (1974). At LT, LA concentration was also significantly higher during 1-legged exercise. The reason for higher ($p < 0.05$) lactates during 1- vs 2-legged work may be explained by the increased glycogen utilization (Stamford et al., 1978a) with 1-legged exercise. Other research has stated that LA generated during low-intensity exercise is catabolized immediately and used as an energy source (Ivy et al., 1980), primarily by ST fibers (Jorfeldt, 1970). Therefore, during 2-legged exercise, LA concentrations should be lower at a given submaximal power output than 1-leg because of an increased aerobic muscle mass oxidizing the accumulating LA and decreased glycolytic motor units generating it.

Variables at VO_2 max are in agreement with previous research. The VO_2 ratio between one- and two-legs was 0.74-0.85 (Saltin et al., 1976; Davies and Sargeant, 1974; Stamford et al., 1978b). Because 1-legged VO_2 max was able to reach 74-85% of 2-legs, but yet only half the muscle mass was utilized, this suggests that it is not the active muscle which limits VO_2 max but rather the central support for the muscle; otherwise the 2-legged VO_2 max should have been twice the 1-legged value.

In summary, this study suggests that O_2 cost is related to the VT and LT and that the central circulation limits VO_2 max during 2-legged exercise.

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Table 1. Physical characteristics of the subjects (n=10)

Subject	Age (yr)	Height (cm)	Weight (kg)	VO_2 max ($l\ min^{-1}$) 1-legged	VO_2 max ($l\ min^{-1}$) 2-legged
1	23	178.0	80.0	3.43	4.00
2	21	179.5	70.9	3.36	4.32
3	25	179.0	89.3	3.90	5.21
4	18	176.0	69.6	3.48	4.40
5	20	177.4	79.5	3.25	4.13
6	21	174.0	72.5	2.59	3.58
7	25	175.5	74.6	3.40	4.38
8	21	180.0	74.2	3.23	4.10
9	21	173.0	77.8	3.46	4.10
10	21	176.5	70.7	3.45	4.45
Mean	21.6	176.8	75.9	3.36	4.27
+ SE	0.7	0.7	1.9	0.10	0.13

Figure I - The exercise testing schedule for one- and two- legged VO_2 max and thresholds tests

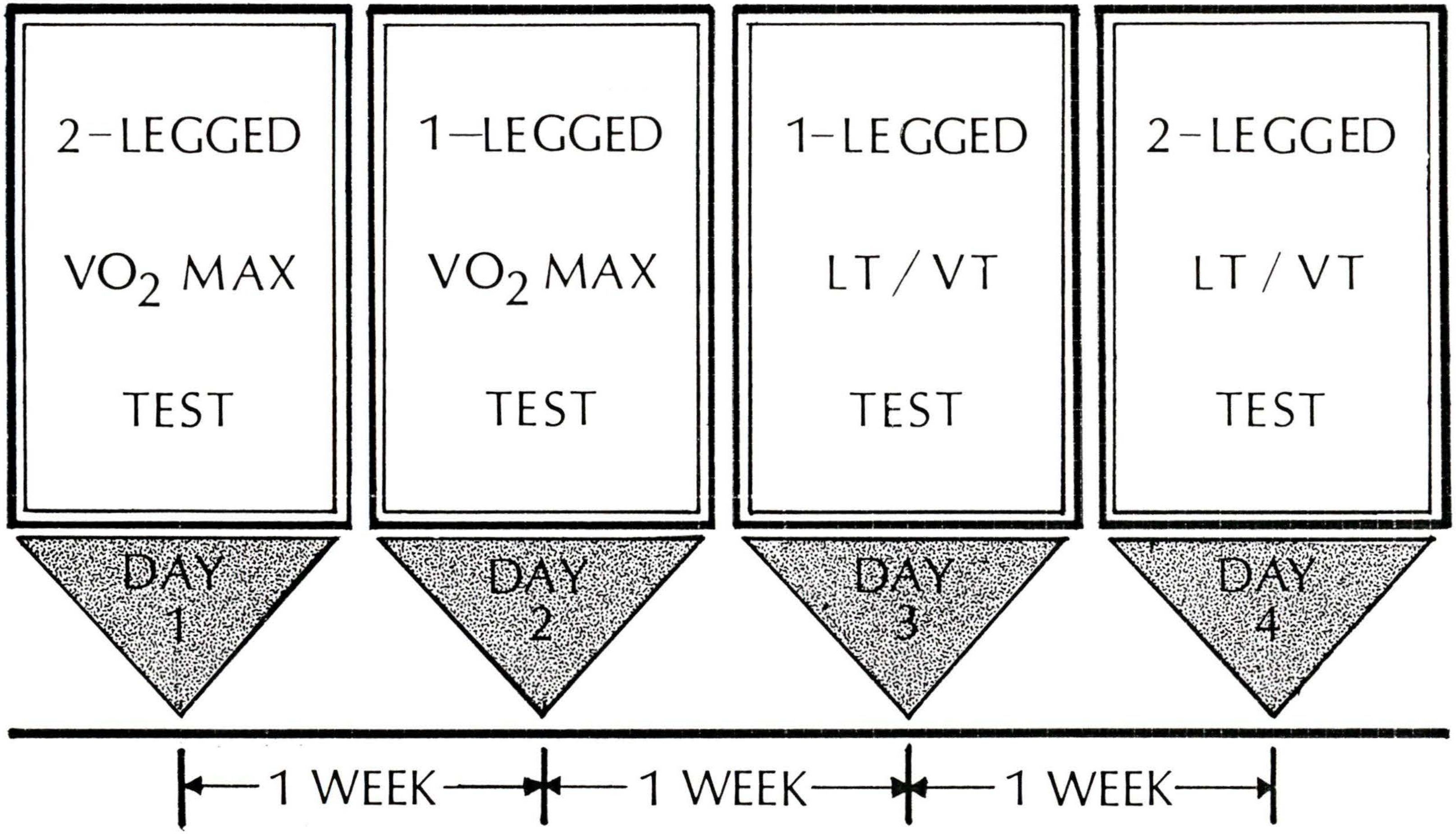

2-LEGGED
VO_2 MAX
TEST
1-LEGGED
VO_2 MAX
TEST
1-LEGGED
LT / VT
TEST
2-LEGGED
LT / VT
TEST
DAY 1
DAY 2
DAY 3
DAY 4
1 WEEK
1 WEEK
1 WEEK

Table 2. Physiological responses of 1- vs. 2-legged cycling at the lactate and ventilation threshold.

	1-legged (n=10)		2-legged (n=8)	
	LT	VT	LT	VT
Lactate	2.62^a		1.97^a	
(mmol l^{-1})	0.16		0.07	
VO_2	1.99	2.15	2.21	2.15
(l min^{-1})	0.11	0.08	0.13	0.18
V_E		60.60		55.40
(l min^{-1})		3.35		4.67
HR (bpm)	135.0	139.0	143.0	139.0
	7.8	8.0	4.6	4.2
R	0.99	1.00	1.02	1.01
	0.03	0.01	0.02	0.02

Values are means + SE

Paired letters (aa) indicate significance at $p < 0.05$

Table 3. Physiological responses of 1- and 2-legged exercise at varying workloads.

	1-leg (n=10)			2-legs (n=8)		
	50 W	100 W	150 W	50 W	100 W	150 W
Lactate	1.80	2.40^a	4.34^b	1.57	1.68^a	1.82^b
(mmol l^{-1})	0.10	0.16	0.33	0.11	0.06	0.08
VO_2	1.08^a	1.68^b	2.61^c	0.82^a	1.18^b	1.63^c
(l min^{-1})	0.02	0.01	0.06	0.13	0.17	0.14
V_E	31.98^a	47.94^b	83.60^c	24.80^a	31.79^b	42.77^c
(l min^{-1})	0.66	0.75	1.71	2.30	2.20	2.76
R	0.96	0.97^a	1.05^b	0.91	0.89^a	0.97^b
	0.06	0.03	0.04	0.06	0.06	0.05
HR (bpm)	92.0	119.0^a	151.0^b	88.0	100.0^a	116.0^b
	4.9	5.6	6.7	4.4	5.0	5.1

Values are means + SE

Paired letters indicate significance at $p < 0.05$

Table 4. Maximal performance values for oxygen consumption (VO_2), ventilation (V_E), and heart rate (HR).

	1-leg			2-legs		
	VO_2 max (l min^{-1})	V_E max (l min^{-1})	HR max (bpm)	VO_2 max (l min^{-1})	V_E max (l min^{-1})	HR max (bpm)
Mean	3.36^a	145.60^b	179.0^c	4.27^a	169.94^b	195.0^c
+SE	0.10	8.51	3.5	0.13	9.30	2.7

Paired letters indicate significance at $p < 0.05$

Figure 2 - Mean oxygen consumption (VO_2; 1 min^{-1}) responses during incremental power outputs (W) for one- and two-legged cycling

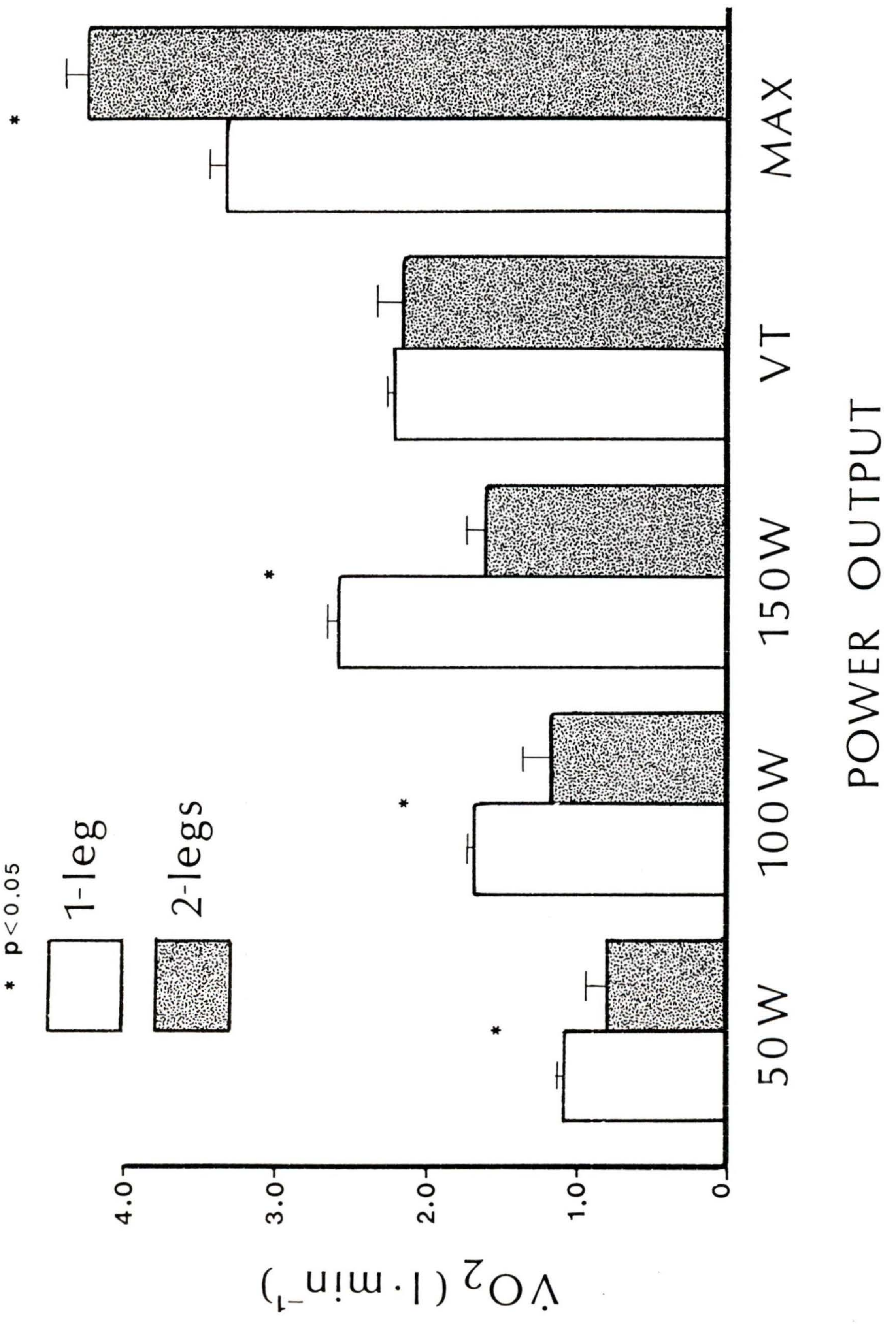

* p < 0.05
1-leg
2-legs
V̇O2 (l · min⁻¹)
0
1.0
2.0
3.0
4.0
50W
100W
150W
VT
MAX
POWER OUTPUT
*

Figure 3 - Mean ventilation (V_E; l min^{-1}) responses during incremental power outputs (W) for one- and two-legged cycling

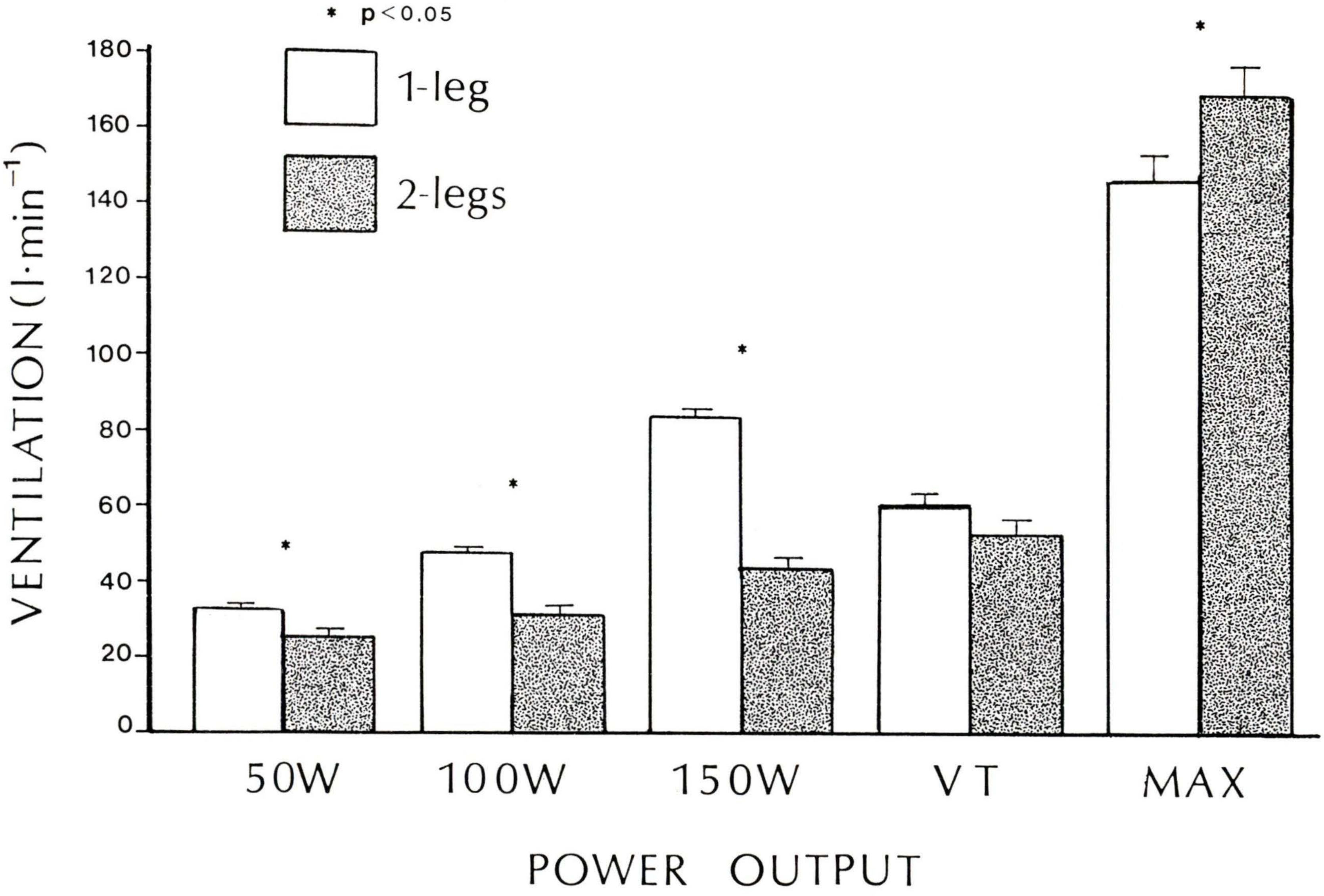

* p<0.05
1-leg
2-legs
VENTILATION (l·min⁻¹)
180
160
140
120
100
80
60
40
20
0
*
*
*
*
50W
100W
150W
VT
MAX
POWER OUTPUT

Figure 4 - Mean lactate (LA; mmol l^{-1}) responses during incremental power outputs (W) during one- and two-legged cycling

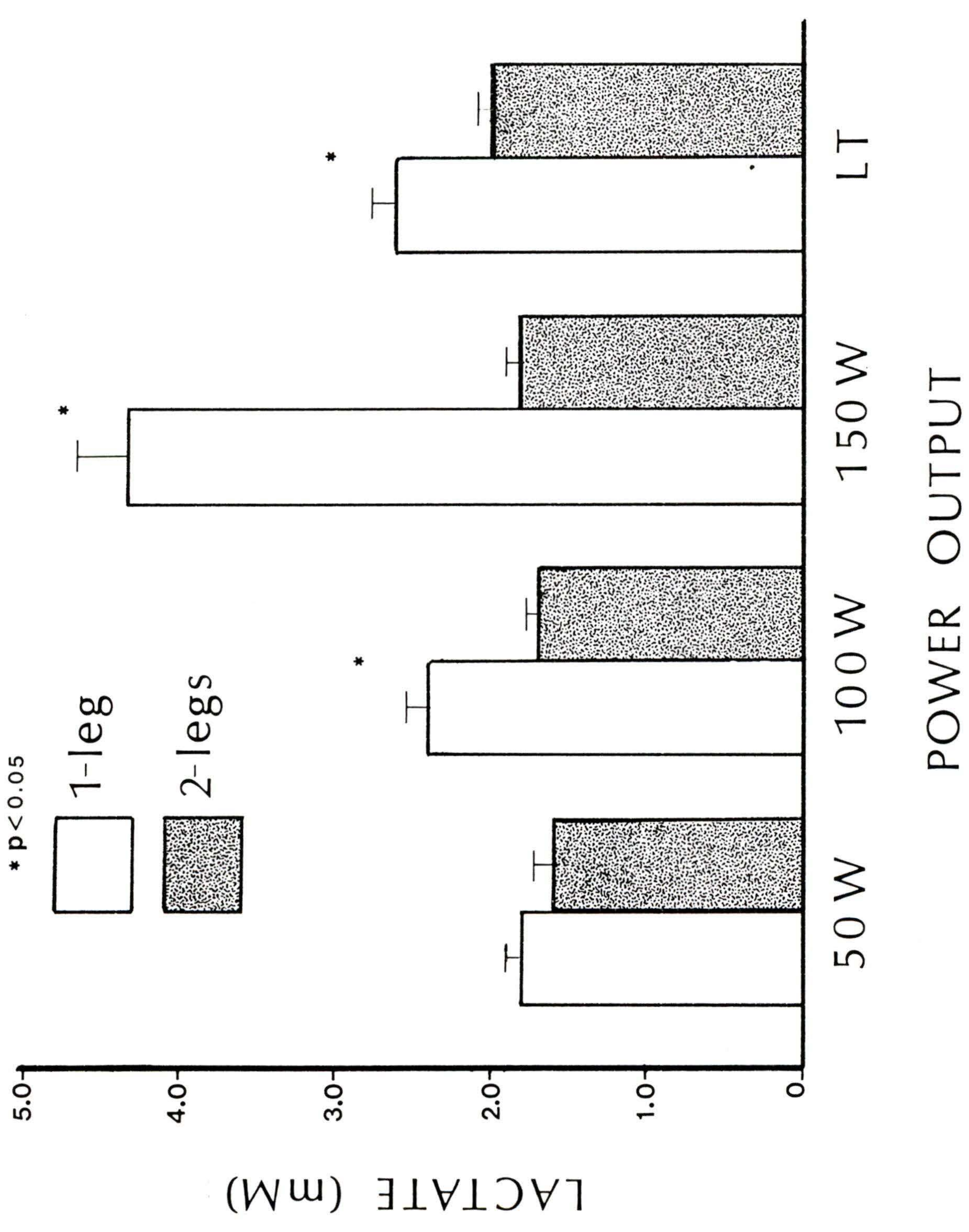
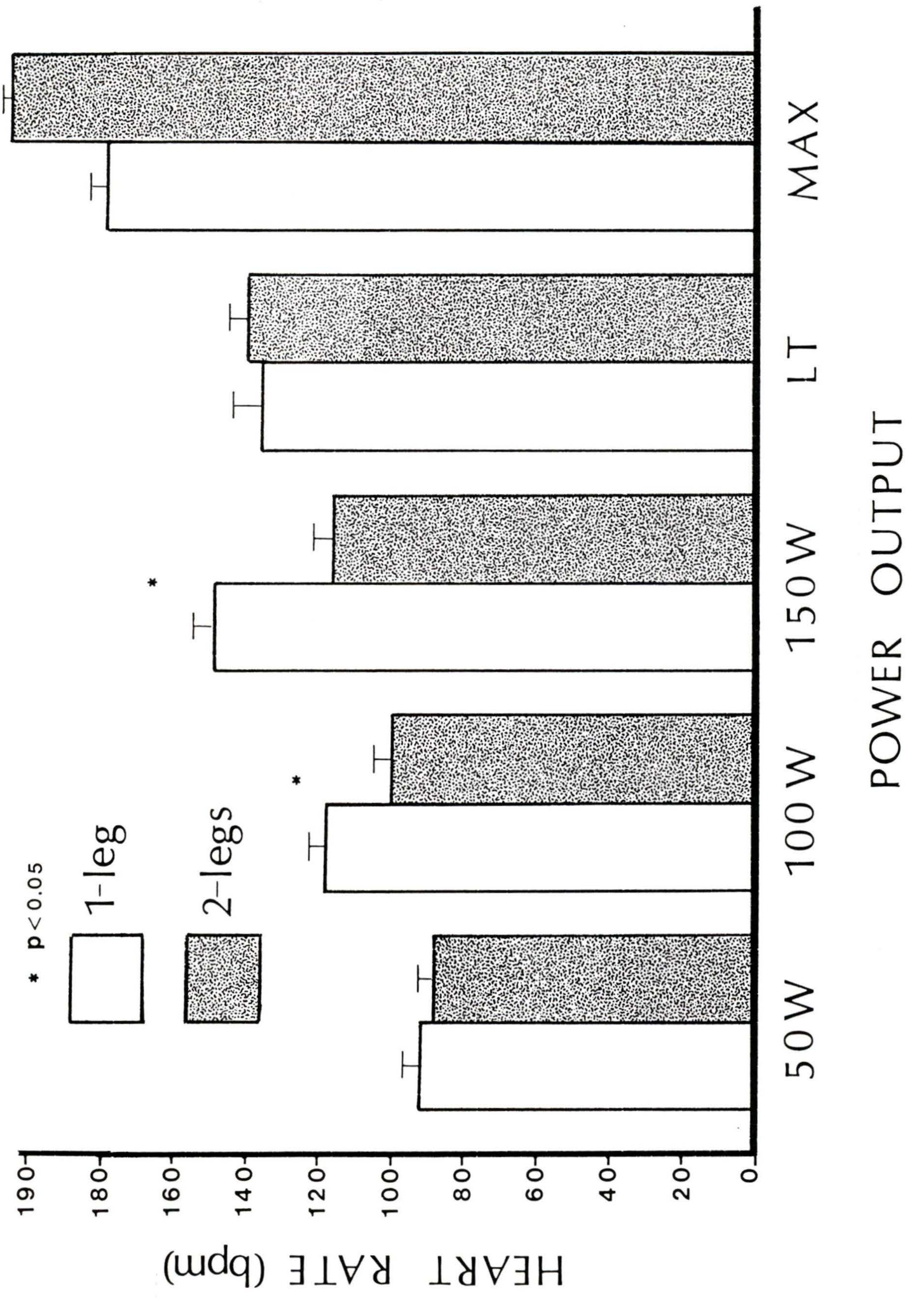

* p < 0.05
1-leg
2-legs
LACTATE (mM)
5.0
4.0
3.0
2.0
1.0
0
*
*
*
50 W
100 W
150 W
LT
POWER OUTPUT

Figure 5 - Mean heart rate (bpm) responses during incremental power outputs (W) for one- and two-legged cycling


HEART RATE (bpm)
190
180
160
140
120
100
80
60
40
20
0
* p < 0.05
1-leg
2-legs
*
*
*
50W
100W
150W
LT
MAX
POWER OUTPUT

CHAPTER III

THE EFFECTS OF PRIOR EXERCISE ON THE LACTATE AND VENTILATORY THRESHOLD

Abstract

This study examined the effects of prior exercise on the lactate (LT) and ventilation (VT) thresholds. Ten healthy male subjects (VO_2 max= 4.27 l min^{-1}) volunteered to perform one-legged cycling. Muscle glycogen reduction was achieved by cycling at 75-85% of maximal heart rate for 60-75 min, and through a low carbohydrate diet. Pre- and post-exercise threshold tests employed a 3 min continuous protocol in 16 Watt (W) increments. Muscle biopsies were taken from the vastus lateralis before the prior exercise (PE) ride, the post-threshold test, and before testing the non-exercised (NE) leg. An I.V. catheter placed in the antecubital vein was used for serial blood samples taken at rest, and during the final 30 s of each progressive load. Gas analysis was calculated every 30 s (Beckman Metabolic Measurement Cart). Biopsies showed that the PE and diet regimen reduced ($p< 0.05$) muscle glycogen in the PE leg (46.7%), and NE leg (36.4%). R and venous lactate (LA) concentrations were reduced ($p< 0.05$) at LT and VT in both the PE and NE leg. VO_2 at a blood LA concentration of 4 mmol l^{-1} was higher ($p=0.07$) in the PE leg at LT (2.89 vs. 2.46 l min^{-1}), but no difference existed between pre- and post- conditions in the NE leg at LT. These results suggest that LA accumulation at LT and VT are altered by endurance exercise performed 24hr prior to testing, and that using an arbitrary blood LA concentration of 4 mmol l^{-1} as a criterion for the LT is not warranted.

Introduction

Endurance has been defined as the 'ability to repeat a given movement' (Edington and Edgerton, 1976). Since aerobic energy supply is fatigue resistant, endurance training generally implies training the aerobic metabolic pathways and the physiological systems responsible for oxygen transport.

In order to provide a comprehensive profile of the endurance athlete it has been proposed that an important measurement is the 'anaerobic threshold' (AT) (MacDougall and Sale, 1981). Wasserman et al. (1973) originally defined AT as a non-linear breakaway in V_E vs. VO_2. This has been demonstrated to occur at approximately the same time as an increase in plasma lactate (LA) (Davis et al., 1976). The AT has also been defined as the power output during progressive steady state exercise where blood LA begins to accumulate significantly above resting levels (2-4 mmol l^{-1}) (MacDougall, 1977; Kindermann et al., 1979). Therefore, the AT has two distinct components: a lactate (LT) and ventilation (VT) threshold.

A review of the literature reveals there has been a number of problems associated with the anaerobic threshold. One such problem is an appropriate definition to define threshold. Researchers have used such terminology as lactate threshold (LT) (Hughes, et al., 1982) ventilation threshold (VT) (Brooks and Fahey, 1984), proportional limit (Jones and Ehrsam, 1982), onset of blood lactate accumulation (OBLA) (Sjodin et al., 1982), aerobic threshold (AerT) (Skinner & McLellan, 980), anaerobic threshold (AT) (Wasserman et al., 1973; Davis et al., 1976), threshold of decompensated metabolic acidosis (TDMA) (Reinhard et al., 1979), lactate (LAT), and ventilation anaerobic threshold (VAT) (Green et al., 1983), respiratory compensation threshold (RCT) (Simon, et al., 1983), and hyperventilation threshold (HVT) (Scheen et al., 1981), to reflect the metabolic acidosis due to muscle lactate production.

A prerequisite for prolonged endurance exercise at the highest possible intensity is that lactate does not accumulate in muscle (Komi et al., 1981). Thus, the ability of an athlete to perform endurance activity is directly related to his 'anaerobic threshold' (MacDougall & Sale, 1981) Therefore, we must understand the factors which affect its measurement.

Recently, Heigenhauser et al. (1983) indicated that prolonged endurance activity, performed 24 hr prior to testing, altered selected ventilatory and respiratory measures (i.e. VO_2, HR, V_E, VCO_2) at any given power output. In addition, both venous LA and R were significantly lower which suggests that the lactate and ventilatory thresholds are altered by prior endurance exercise. Thus, the intent of this study is to confirm the effects on the lactate and ventilation thresholds of prolonged endurance exercise on the day prior to a test, and to determine whether the locus of the effect is in the peripheral muscle or in the central circulation.

Method

Subjects

Since the first purpose of this study is to determine if prior endurance exercise will alter the measurement of the LT and VT, ten healthy male subjects (mean 2-legged VO_2 max= 4.27 l min^{-1}; 50-65 ml kg^{-1} min^{-1}) attending the University of Victoria volunteered to perform endurance exercise on a cycle ergometer. Informed consent was obtained and a PAR Q (Physical Activity Readiness Questionnaire) was completed. Physcial characteristics of the subjects are presented in Table 1. Of the ten subjects, seven were members of various university varsity teams.

Since the second purpose of this study is to determine whether prior exercise affects the thresholds through a central (circulatory) or peripheral influence, one-legged cycling was performed for both the prior exercise and the measurement of LT and VT.

Procedures

The exercise and testing schedule is presented in Fig 1. The PE ride performed on Day 3 involved cycling (Monark cycle ergometer) for approximately 60-75 min at an intensity of 75-85% of maximal heart rate achieved during a 1-legged VO_2 max pre-test. The PE ride was followed by one minute repeated sprint bouts (4-6) (1:1 work-rest ratio) at 100% of the power output at VO_2 max. To maintain low muscle glycogen, a low carbohydrate diet (mean= 2400 calories) consisting of 50% Protein, 40% Fats, 10% Carbohydrates was adhered to immediately following the PE ride until all testing was completed 30 hr later.

The 1-legged submaximal (85-90% VO_2 max) threshold tests performed on Days 1, 2, and 4 consisted of a 3 min continuous protocol on an electrically braked cycle ergometer (Quinton Instruments, model 844). The protocol included a warm-up at 50 W, after which time the load was increased to 82 W and then 16 W every load thereafter.

A pedalling frequency of 60-70 rpm was maintained throughout the test duration of 15-24 minutes. All subjects were asked to refrain from their regular physical activity the day prior to the PE and threshold rides. Metabolic and respiratory measures (R, V_E, VO_2, VCO_2, FE_{CO2}, FE_{O2}) were monitored every 30 s (Beckman Metabolic Measurement Cart). Standard gases of known concentrations were used to calibrate the gas analyzers before each test.

In order to accommodate 1-legged cycling at higher loads, toe clips and a heel strap were used to secure the foot. The NE leg was rested on the cycle ergometer between the pedals.

An indwelling catheter (Deseret Angio-Cath, 20 g x 3.2 cm), placed in the antecubital vein was used to take samples at rest and during the final 30 s of each load throughout the test. Blood samples (0.50 ml) were immediately deproteinized in 2.0 ml of ice cold 4% perchloric acid, centrifuged, and then frozen for subsequent spectrophotometric analysis (Sigma Lactate Kit; Sigma Chemical Company, 1981).

Muscle biopsy samples were taken from the vastus lateralis muscle (N=3) before the PE ride on Day 3, and immediately before the threshold test, 24 hr later, on Day 4. A third sample was also taken from the NE leg before testing in the afternoon of Day 4 (Fig 1). All muscle biopsies were immediately frozen in liquid nitrogen and later analyzed for glycogen employing the glucose-6-phosphatase technique (Bergstrom, 1962).

The criteria for determination of LT and VT was a nonlinear increase in LA vs. VO_2 and V_E vs. VO_2, respectively. A linear regression model was used to determine 'breakaway' for each individual test. Individual LT and VT were determined and group means calculated. A Student's t-test was used to determine statistical differences between tests on Days 1 and 2, and between the pre- and post- PE tests.

Results

Muscle biopsy samples substantiate that the exercise and diet regimen significantly ($p< 0.01$) reduced (46.7%) muscle glycogen content of the PE leg from 126.3 + 5.3 umol g^{-1} ww at rest to 67.4 + 11.9 umol g^{-1} ww before the exercise test 24 hr later. The NE leg showed a 36.4% reduction in glycogen stores (Table 2). No significant differences ($p< 0.05$) existed between threshold tests on Day 1 and Day 2 performed one week apart under identical experimental conditions (Table 4).

Venous LA concentration increased curvilinearly with increased VO_2 (l min^{-1}) (Fig 2). Lactate levels, however, were significantly ($p< 0.05$) lower under reduced muscle glycogen conditions. At LT, venous LA concentrations were 2.66 and 1.99 mmol l^{-1} for the PE leg, pre- and post-, respectively. At VT, LA concentrations were 3.35 and 2.56 mmol l^{-1}, respectively. The NE leg was also significantly lower in LA at LT (2.87 vs. 2.26 mmol l^{-1}) and VT (3.59 vs. 2.74 mmol l^{-1}), pre- and post-, respectively. Reduced muscle glycogen did not affect VO_2 (l min^{-1}) or VE at the LT and VT among threshold tests, although R was significantly lower for both the exercise and non-exercised legs (Table 3). LT occurred at 62.2% VO_2 max under both NE and PE conditions, while VT occurred at 64.3% and 66.4% VO_2 max, respectively. HR increased linearly with each load under both conditions but no significant differences were observed at any power output.

Oxygen consumption at a blood LA concentration of 4 mmol was higher (p=0.07) in the PE leg. VO_2 was 2.46 l min^{-1} under normal conditions and 2.89 l min^{-1} in the PE leg. No differences were found in the NE leg (Table 5; Fig 2).

The ratio between one-legged and two-legged maximal oxygen uptake ranged between 0.74-0.85.

Discussion

The purpose of this study was to determine if prior exercise affects the LT and VT. If the effect is mediated peripherally it will be demonstrated only when the prior exercised leg is tested. If it is centrally mediated it will occur when both the prior exercised and non-exercised legs are tested.

Blood LA concentration was significantly ($p < 0.05$) reduced at the LT and VT when both the PE and NE legs were tested. However, VO_2 ($l\ min^{-1}$) at these thresholds was not affected. These results are consistant with Ivy et al. (1981) who demonstrated that VO_2 was not altered by changes in substrate availability. Oxygen consumption, however, at a blood LA concentration of 4 mmol l^{-1} was higher ($p=0.07$) after the prior exercise test (Fig 2). This has implications for the measurement of LT and VT. If VO_2 at LT or VT is used, prior exercise has no effect. However, if lactate levels at LT or VT, or a LA concentration of 4 mmol l^{-1} are employed as the criteria, then prior exercise will affect their measurement. Therefore, BLA4mM is not justifiable as a criterion to indicate the lactate threshold. Because muscle glycogen and LA levels were significantly reduced in both the PE and NE leg, it implies the effect was mediated centrally rather than when the prior exercise muscles were tested. This reduced glycogen content of non-exercising muscle mass has also been reported by Bonen (1983). In addition, Karlsson et al. (1975) have illustrated that 'non-exercising' muscles become metabolically affected because of heavy prior exercise.

These data support results of Saltin et al. (1976) who used a 1-legged model to establish differences between local and general effects of training. Their results showed that the non-trained leg also improved, suggesting a 'close interplay between local and central factors' are responsible for metabolic changes resulting from the training program.

In summary, endurance exercise performed 24 hr prior to the measurement of the LT or VT will affect venous LA concentration and VO_2 at a lactate level of 4 mmol l^{-1}. The effect of preliminary exercise is mediated in both prior exercised and non-exercised legs which suggest central (circulatory) factors play a significant role in this response.

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Table 1. Physical characteristics of the subjects (n=10)

Subject	Age (yr)	Height (cm)	Weight (kg)	VO_2 max (l min^{-1})	
				1-legged	2-legged
1	23	178.0	80.0	3.43	4.00
2	21	179.5	70.9	3.36	4.32
3	25	179.0	89.3	3.90	5.21
4	18	176.0	69.6	3.48	4.40
5	20	177.4	79.5	3.25	4.13
6	21	174.0	72.5	2.59	3.58
7	25	175.5	74.6	3.40	4.38
8	21	180.0	74.2	3.23	4.10
9	21	173.0	77.8	3.46	4.10
10	21	176.5	70.7	3.45	4.45
Mean	21.6	176.8	75.9	3.36	4.27
+ SE	0.7	0.7	1.9	0.10	0.13

Figure 1 - The exercise and testing schedule

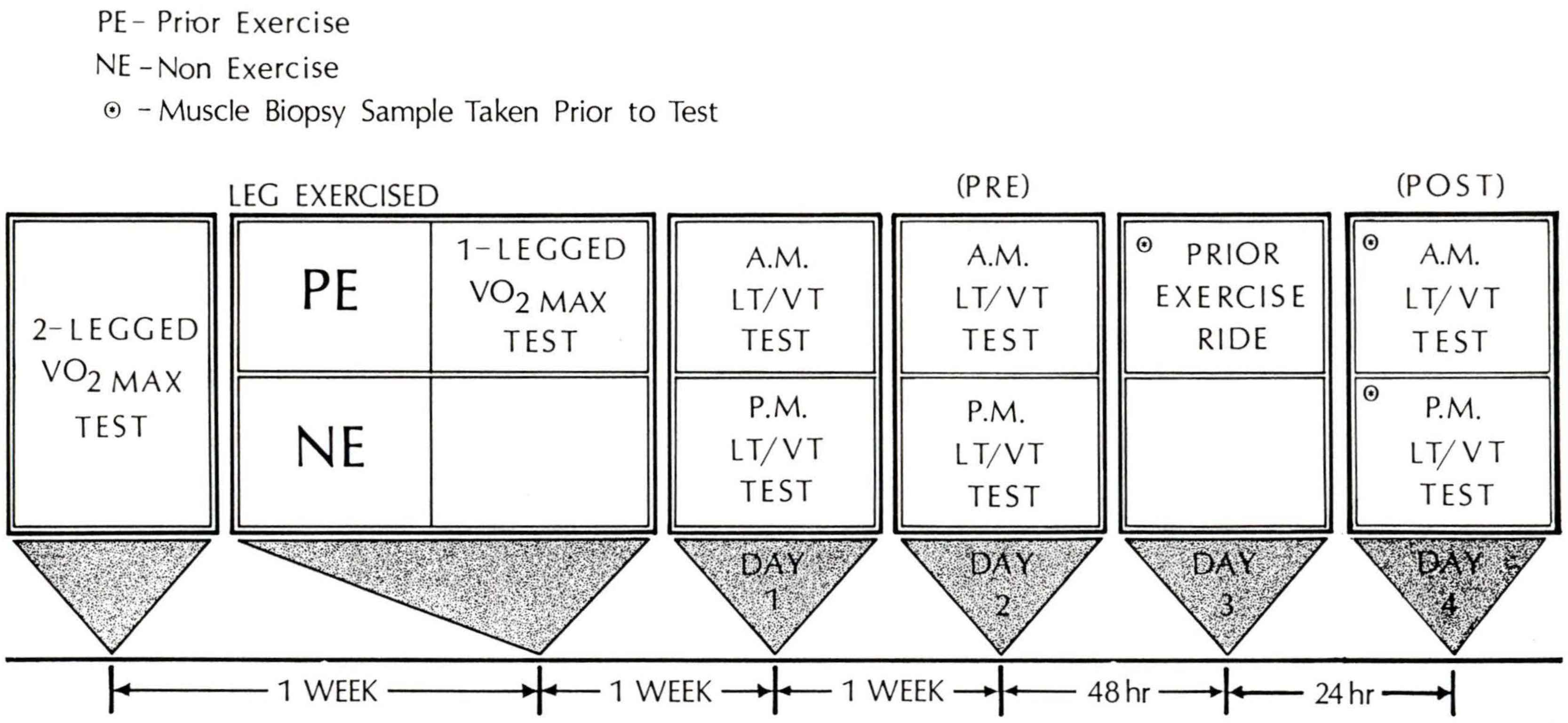
PE- Prior Exercise
NE -Non Exercise
⊙ - Muscle Biopsy Sample Taken Prior to Test
LEG EXERCISED
(PRE)
(POST)
2-LEGGED VO2 MAX TEST
PE
NE
1-LEGGED VO2 MAX TEST
A.M. LT/VT TEST
P.M. LT/VT TEST
A.M. LT/VT TEST
P.M. LT/VT TEST
⊙ PRIOR EXERCISE RIDE
⊙ A.M. LT/VT TEST
⊙ P.M. LT/VT TEST
DAY 1
DAY 2
DAY 3
DAY 4
1 WEEK
1 WEEK
1 WEEK
48 hr
24 hr

Table 2. Glycogen content of muscle biopsy samples (n=3)

Subject	Rest	PE-Leg (24 hr later)	NE- Leg (30 hr later)
1	117.4	53.2	62.3
5	135.7	91.1	100.2
2	125.7	57.9	78.3
Mean	126.3	67.4^*	80.4^+
+ SE	5.3	11.9	11.0

Values are means + SE in umol g^{-1} wet weight

* Significantly different at $p < 0.01$

\+ Significantly different at $p < 0.05$

Table 3. Physiological responses of one-legged cycling for the prior exercised (PE) and non-exercised (NE) legs at lactate (LT) and ventilatory thresholds (VT) for pre- and post- glycogen reduction

	PE				NE			
	Pre		Post		Pre		Post	
	LT	VT	LT	VT	LT	VT	LT	VT
LA	2.62^{a}	3.35^{b}	1.99^{a}	2.56^{b}	2.87^{cd}	3.59^{ce}	2.26^{d}	2.74^{e}
mmol l^{-1}	0.16	0.30	0.06	0.18	0.24	0.26	0.21	0.26
VO_2	1.99	2.15	2.09	2.23	2.20	2.24	2.06	2.15
l min^{-1}	0.11	0.08	0.07	0.06	0.10	0.06	0.06	0.06
V_E	60.00	60.60	58.00	58.70	61.00	64.20	59.50	60.40
l min^{-1}	4.47	3.35	2.15	1.79	2.07	2.30	2.15	2.47
HR	135.0	139.0	138.0	143.0	144.0	145.0	138.0	137.0
	7.8	8.0	6.4	8.1	7.3	8.8	7.4	7.8
R	0.99^{a}	1.00^{b}	0.94^{a}	0.96^{b}	1.06^{c}	1.04^{d}	0.97^{c}	0.98^{d}
	0.03	0.01	0.01	0.02	0.01	0.02	0.01	0.01

Values are means + SE

Paired letters indicate significant differences between tests ($p < 0.05$)

Table 4. Physiological responses to threshold tests performed on Days 1 and 2 under identical experimental conditions

	Day 1		Day 2	
	LT	VT	LT	VT
LA (mmol l^{-1})	2.80	3.35	2.62	3.35
	0.19	0.38	0.16	0.30
VO_2 (l min^{-1})	2.09	2.29	1.99	2.16
	0.07	0.92	0.11	0.78
V_E (l min^{-1})	59.50	63.90	60.00	60.60
	2.15	4.24	4.47	3.35
HR (bpm)	133.00	143.00	135.00	139.00
	7.20	8.10	7.80	8.00
R	1.01	1.01	0.99	1.00
	0.02	0.01	0.01	0.01

Values are means + SE

Table 5. Oxygen consumption at a lactate concentration of 4 mmol during prior exercised and non-exercised conditions

Leg Tested	Prior Exercised		Non-Exercised	
	Pre	Post	Pre	Post
VO_2	2.46	2.89	2.42	2.50
(l min^{-1})	0.13	0.18	0.09	0.13

Values are means + SE

Figure 2a - Pre- and post- venous lactate responses during one-legged cycling under prior exercise conditions

Figure 2b - Pre- and post- venous lactate responses during one-legged cycling under non-exercise conditions

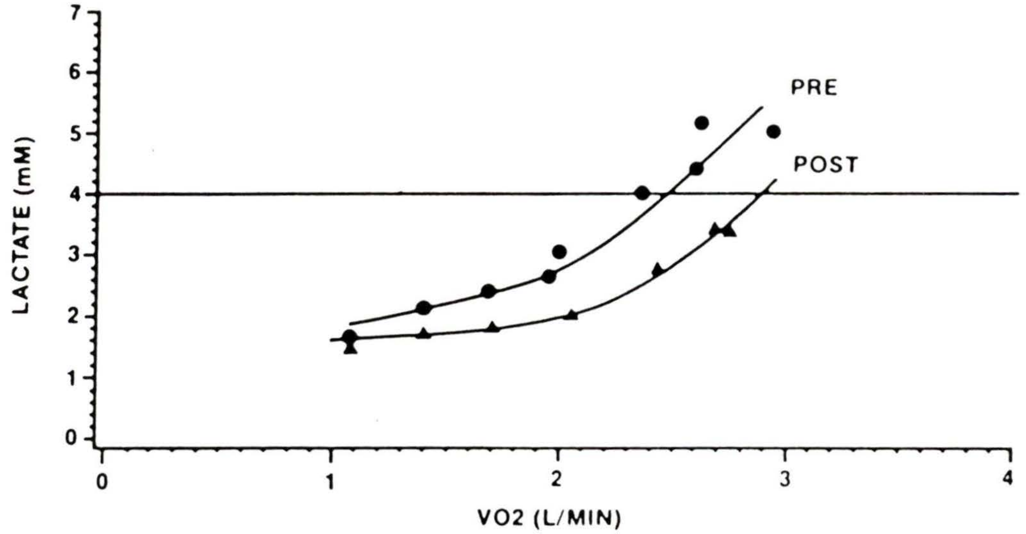
LACTATE (mM)
0
1
2
3
4
5
6
7
0
1
2
3
4
VO2 (L/MIN)
PRE
POST

Fig. 2a

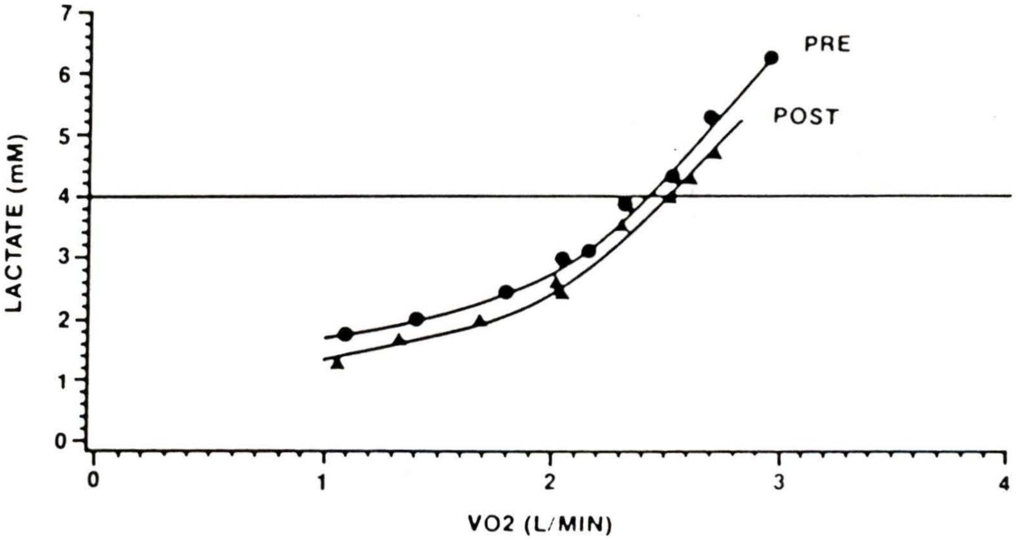
LACTATE (mM)
0
1
2
3
4
5
6
7
0
1
2
3
4
VO2 (L/MIN)
PRE
POST

Fig. 2b

CHAPTER IV

THE RELATIONSHIP BETWEEN LACTATE AND VENTILATORY THRESHOLD: COINCIDENTAL OR CAUSE AND EFFECT?

Abstract

To determine if blood lactate (LA) is the stimulus responsible for 'breakaway' ventilation (V_E), the lactate (LT) and ventilation (VT) thresholds were monitored during one-legged cycling exercise. Ten healthy volunteer male subjects (mean 2-legged VO_2 max=4.27 l min^{-1}) performed prior exercise (PE) to reduce muscle glycogen stores by cycling at 75-85% of maximal heart rate (HR max) for 60-75 min, followed by a 30 hr low carbohydrate diet. Pre- and post- LT and VT tests were performed on a cycle ergometer employing a continuous protocol with increments of 16 W every 3 min. Muscle biopsies were taken from the vastus lateralis muscle before the PE ride, prior to the threshold test 24 hr later, and before testing the non-exercised (NE) leg. An I.V. catheter placed in the antecubital vein was used for serial blood samples taken at rest, and during the final 30 s of each progressive load. Gas analysis was calculated every 30 s (Beckman Metabolic Measurement Cart). Biopsies (N=3) showed that the exercise and diet regimen elicited glycogen reduction which significantly ($p < 0.05$) reduced R and the blood LA concentration in both the PE (2.62 to 1.99 mmol l^{-1}) and NE (2.87 to 2.26 mmol l^{-1}) legs at LT. At VT, LA concentrations were also significantly reduced in the PE (3.35 to 2.56 mmol l^{-1}) and NE (3.59 to 2.77 mmol l^{-1}) legs. VO_2 and V_E, however, were similar between pre- and post-tests. Results of this study suggest that plasma LA accumu-

lation is not responsible for 'breakaway' ventilation during progressive exercise and that LT and VT are not a cause and effect relationship.

Introduction

The concept of anaerobic threshold (AT) was originally advanced by Wasserman, et al. (1973) in an attempt to characterize and explain muscle acidosis. They demonstrated that a disproportionate increase in ventilation (V_E) during progressive exercise was associated with an increase in hydrogen ion concentration $[H^+]$ and P_{CO2} in blood. It has been demonstrated that H^+ and lactate production are stoichiometric. Therefore, in most situations the increased accumulation of lactate in plasma is accompanied by H^+, and an increase in the expired volume of CO_2, and a simultaneous breakaway in V_E (Jones and Ehrsam, 1982). Thus, Wasserman and co-workers (1973) have proposed that this relationship between lactate accumulation and H^+ efflux allows us to employ the ventilation threshold (VT) as an index of the lactate threshold (LT), and they further suggest a causal link between LT and VT. Other researchers have also illustrated that breakaway ventilation generally appears at the same time as an increase in plasma lactate (Davis et al., 1976; Reinhard et al., 1979).

There are, however, a number of factors which can affect the measurement and reproducibility of LT and VT. Studies have demonstrated that such factors include: muscle glycogen depletion and pedaling speed (Hughes et al., 1982); infusion of lactate, glucose and pyruvate in exercising rats (Bagby et al., 1979); the effects of exercise on muscle with or without prior exhaustive work (Karlsson et al., 1975); substrate availability (Ivy et al., 1981); and the effects of reduced muscle glycogen on the ventilation and metabolic responses to exercise, performed 24 hr prior to testing (Heigenhauser et al., 1983). Such factors result in changes in either or both the LT and VT.

Recent studies by Davis and Gass (1981), and Hagberg et al. (1982) have provided evidence that the relationship between LT and VT may only be coincidental. Davis and Gass used two exhaustive exercise tests to induce different blood LA concentrations and demonstrated that VT occurred at the same power output in both tests although lactate levels were different. Also, Hagberg et al. (1982) performed graded exercise on patients with McArdles disease syndrome (a genetic lack of muscle phosphorylase), and showed that a VT occurs without an accumulation of LA or $[H^+]$ in plasma.

Therefore, in order to further investigate the supposed cause and effect relationship between LT and VT, this study was designed to induce a lowered lactate production at a given power output by reducing muscle glycogen through preliminary exercise.

Methods

Subjects

Ten healthy volunteer male subjects (mean 2-legged VO_2 max = 4.27 l min^{-1}; 50-65 ml kg^{-1} min^{-1}) signed informed consent and completed a PAR Q (Physical Activity Readiness Questionnaire) before testing. Physical characteristics of the subjects are presented in Table 1. Of the ten subjects, seven were members of various university varsity teams.

Procedures

Since the purpose of this study was to determine whether a causal link exists between the LT and VT, a one-legged cycling model was employed to compare results between the prior exercised (PE) and non-exercised (NE) leg. The exercise and testing schedule is presented in Fig 1. Initially, all subjects reported to the laboratory for familiarization with the equipment, the nature of the study, and with one-legged cycling. The PE and NE leg were randomly selected to avoid any 'leggedness'.

The LT and VT tests performed on Days 1 & 3, followed a continuous protocol with 3 min incremental steps on an electrically braked cycle ergometer (Quinton Instruments, model 844). The protocol included a warm-up at 50 W and then 16 W increments thereafter. Pedalling frequency was maintained at 60-70 revolutions per minute (rpm) for the 15-24 min test duration. All subjects were asked to refrain from their regular physical activity the day prior to the glycogen reduction and testing days. Metabolic and respiratory measures (R, V_E, VO_2, VCO_2, $FECO_2$, FEO_2) were monitored every 30 s (Beckman Metabolic Measurement Cart). The gas analyzers were calibrated before each test with gases of a known concentration.

In order to accommodate 1-legged cycling at the higher loads, toe clips and a heel strap were used to secure the foot. The non-working leg was rested on the cycle ergometer between the pedals.

Venous blood samples were taken from the antecubital vein with an indwelling catheter (Angio-Cath, 20 g x 3.2 cm) at rest, immediately following the warm-up, and during the final 30 s of each load throughout exercise. The blood samples (0.50 ml) were immediately deproteinized in 2.0 ml of ice cold 4% perchloric acid, centrifuged, and frozen for subsequent lactate analysis (Sigma Lactate Kit; Sigma Chemical Company, 1981).

The PE ride performed on Day 2 involved stationary cycling for approximately 60-75 min at 75-85% of maximal heart rate achieved during a 1-legged VO_2 max pre-test. The PE ride was followed by one minute repeated sprint bouts (4-6) (1:1 work-rest ratio) at VO_2 max. A low carbohydrate diet consisting of 50% Protein, 40% Fats, 10% Carbohydrates (mean= 2400 calories) was adhered to immediately following the PE ride until all testing was completed 30 hr later.

Muscle biopsy samples were taken from the vastus lateralis muscle in three subjects prior to the PE ride on Day 2, and immediately before the threshold test on Day 3. A third sample was then taken from the non-exercised leg before testing in the afternoon (Day 3). All muscle biopsies were immediately frozen in liquid nitrogen and later analyzed for glycogen content employing the glucose-6-phosphatase technique (Bergstrom, 1962).

The criteria for determination of lactate and ventilation thresholds was a non-linear increase in LA vs. VO_2 and V_E vs. VO_2, respectively. A linear regression model was used to determine 'breakaway' for each individual test. Individual lactate and ventilation thresholds (Fig 2) were determined and group means calculated. A Student's t-test was used to determine statistical differences ($p< 0.05$) between pre- and post- tests on Days 1 and 3.

Results

The muscle biopsy samples substantiate that the exercise and diet regimen significantly ($p< 0.05$) reduced the muscle glycogen content of the PE leg (126.3 + 5.3 umol g^{-1} wet weight to 67.4 + 11.9 umol g^{-1} wet weight). However, even though the other leg was rested on the bike during the prolonged exercise ride, this 'non-exercised' leg also showed a significant reduction in glycogen content prior to testing on Day 3 (126.3 + 5.3 umol g^{-1} wet weight to 80.3 + 11.0 umol g^{-1} wet weight) (Table 2). The glycogen reduction was 46.7% and 36.4% for the PE and NE leg, respectively.

Venous lactate (LA) concentration during the pre- and post- tests demonstrated a curvilinear increase with increasing VO_2. Lactate levels, however, were significantly reduced under the prior exercise conditions at both the LT and VT. The lactate concentration was also significantly reduced ($p< 0.05$) in the NE leg at LT and VT between tests (Table 3).

As shown in Fig 2, the LT and VT occurred at approximately the same VO_2, thus the reduced muscle glycogen content did not affect VO_2 at either threshold in the PE leg. A similar trend was also shown in the NE leg in which no significant differences ($p< 0.05$) in VO_2 were found between pre- and post- conditions (Table 3). The LT occurred at 62% VO_2 max both pre- and post- for the PE leg, and at 64% and 66% VO_2 max for the VT pre- and post- , respectively.

Heart rate (HR) demonstrated a linear increase with work load but no significant difference was shown between tests. The R value was significantly lower for both the PE and NE leg at threshold during the post- exercise tests.

Discussion

Research has suggested that during progressive exercise the non-linear increase in V_E is associated with the production of lactic acid and $[H^+]$ and the concommitant rise in the P_{CO2}. The ventilatory response is then mediated via the carotid bodies (Whipp, 1978; Whipp and Davis, 1979; Jones and Ehrsam, 1982).

The present study found that the non-linear 'breakaway' in V_E occurred at the same VO_2 ($l\ min^{-1}$) under prior exercise (PE) and normal exercise conditions. During the PE condition however, LA production was significantly ($p< 0.05$) decreased at all power outputs. Therefore, these data concur with Hagberg et al. (1982) and Davis and Gass (1981) and suggest that venous LA concentration is not primarily responsible for either the ventilatory drive or the ventilation threshold. Collectively, these studies suggest that other stimuli(us) beside LA is responsible for the ventilation threshold. Although $[H^+]$ was not examined in the present investigation, Hagberg et al. (1982) showed that humoral factors such as pH, P_{O2} and P_{CO2} are not the primary stimuli for the V_E breakaway.

Gleim et al. (1984) suggest that the lactate threshold may be a result of sympathetic activity. At the LT, catecholamine and plasma renin activity are elevated. This suggests that sympathetic activity is augmented, which may also stimulate ventilation.

These results illustrate that the LA concentration was significantly different ($p < 0.05$) between pre- and post-tests for both exercise conditions (PE and NE). Since VO_2 ($l\ min^{-1}$) was similar pre- and post-; the O_2 cost was the same at threshold. Therefore, to maintain the power required to perform progressive exercise, FT motor units are recruited at approximately the same time during both conditions. Ivy et al. (1980) revealed that muscle fiber composition is related to the level of VO_2 at 'anaerobic threshold'. Thus, the recruitment of FT motor units could be the reason for both the increased lactate levels and the non-linear increase in V_E via the hypothalamus and medulla. Recent work by Heigenhauser et al. (1983) further suggest that a neurogenic stimulus may be responsible for the ventilation drive during progressive exercise under glycogen reduced conditions, and in animal studies (Koa et al., 1964; Tibes, 1977), increased neural activity has been associated with an increase in V_E.

These results also suggest that FFA oxidation was enhanced during PE conditions because R values were lower and VO_2 remained constant in the reduced state. It is known that increased FFA utilization impedes the rate of glycolysis, therefore lowering LA levels (Hughes et al., 1982; Heigenhauser et al., 1983).

Since previous research has altered LA concentrations and glycogen levels (Davis and Gass, 1981; Hughes et al., 1982) to determine the relationship between the lactate and ventilation threshold; and since a 1-legged model was used by Stamford et al. (1978a) to observe changes at the 'anaerobic threshold'; it was thought that by employing a 1-legged model with reduced LA and glycogen levels, the results would further substantiate the relationship between the LT and VT. Although one leg was thought to be inactive during the one-legged PE ride, the results indicated that glycogen reduction and lower lactate concentrations occurred during the threshold test. VO_2 however, was similar at the 'breakaway' points (LT and VT). Regardless of the condition imposed (PE or NE) and therefore level of LA, it appears that the breakaway in V_E may be related to the O_2 cost (and power output) which triggers motor unit recruitment within the exercising muscle.

Therefore, the present study demonstrates that the accumulation of venous lactate is not responsible for the breakaway in V_E. These results further support the contention that the LT and VT are only coincidental and not a cause and effect relationship (Brooks and Fahey, 1984; Green et al., 1983; Hagberg et al., 1982; Davis and Gass, 1981). Thus, the use of ventilatory measures to imply lactic acidosis during incremental exercise do not seem warranted.

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Table 1. Physical characteristics of the subjects (n=10)

Subject	Age	Height	Weight	VO_2 max ($l\ min^{-1}$)	
	(yr)	(cm)	(kg)	1-legged	2-legged
1	23	178.0	80.0	3.43	4.00
2	21	179.5	70.9	3.36	4.32
3	25	179.0	89.3	3.90	5.21
4	18	176.0	69.6	3.48	4.40
5	20	177.4	79.5	3.25	4.13
6	21	174.0	72.5	2.59	3.58
7	25	175.5	74.6	3.40	4.38
8	21	180.0	74.2	3.23	4.10
9	21	173.0	77.8	3.46	4.10
10	21	176.5	70.7	3.45	4.45
Mean	21.6	176.8	75.9	3.36	4.27
+ SE	0.7	0.7	1.9	0.10	0.13

Figure 1 - The exercise and testing schedule

PE - Prior Exercised
NE - Non Exercised
⊙ - Muscle Biopsy Sample Taken Prior to Test

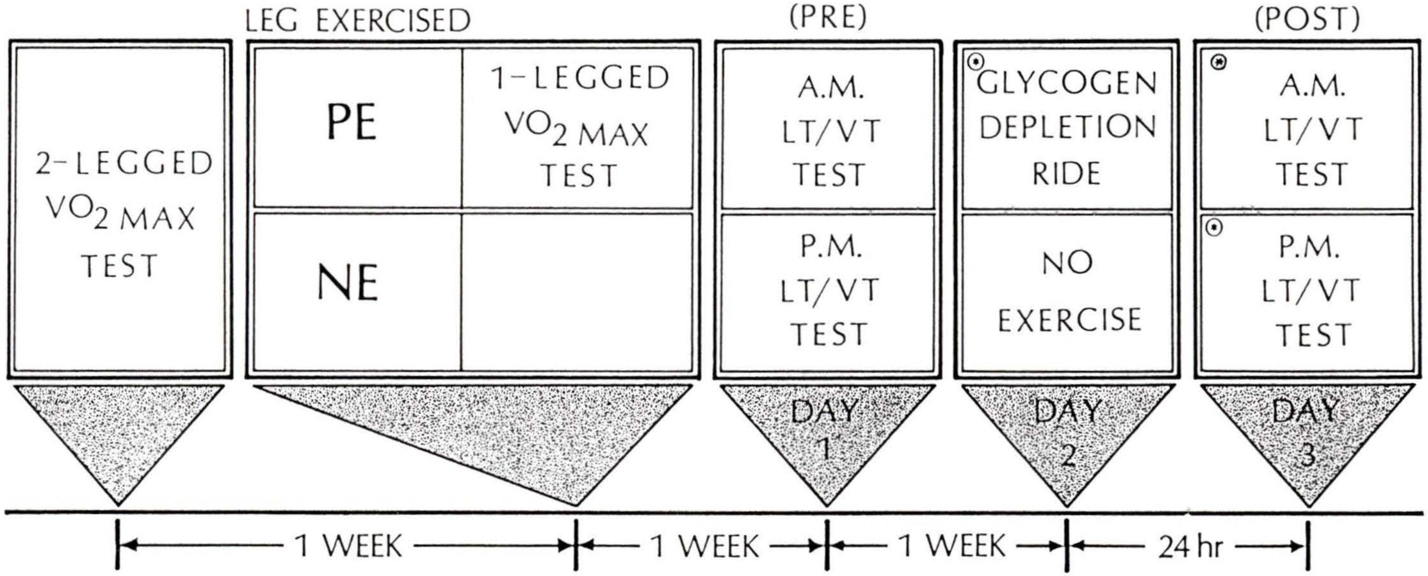


Table 2. Glycogen content of muscle biopsy samples (n=3)

Subject	Rest	PE-Leg (24 hr later)	NE- Leg (30 hr later)
1	117.4	53.2	62.3
5	135.7	91.1	100.2
2	125.7	57.9	78.3
Mean	126.3	67.4*	80.4+
+ SE	5.3	11.9	11.0

Values are means + SE in umol g^{-1} wet weight

* Significantly different at $p< 0.01$

\+ Significantly different at $p< 0.05$

Table 3. Physiological responses of one-legged cycling for the prior exercised (PE) and non-exercised (NE) legs at lactate (LT) and ventilatory thresholds (VT) for pre- and post- glycogen reduction

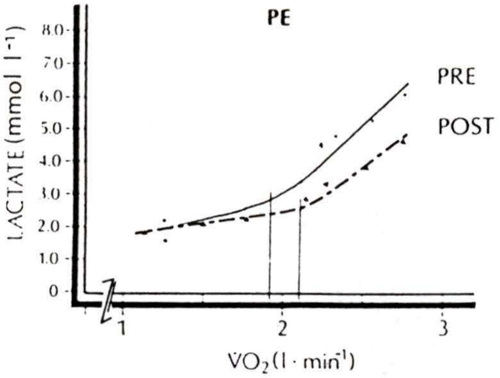
	PE				NE			
	Pre		Post		Pre		Post	
	LT	VT	LT	VT	LT	VT	LT	VT
LA	2.62^{a}	3.35^{b}	1.99^{a}	2.56^{b}	2.87^{cd}	3.59^{ce}	2.26^{d}	2.74^{e}
mmol l^{-1}	0.16	0.30	0.06	0.18	0.24	0.26	0.21	0.26
VO_2	1.99	2.15	2.09	2.23	2.20	2.24	2.06	2.15
l min^{-1}	0.11	0.08	0.07	0.06	0.10	0.06	0.06	0.06
V_E	60.00	60.60	58.00	58.70	61.00	64.20	59.50	60.40
l min^{-1}	4.47	3.35	2.15	1.79	2.07	2.30	2.15	2.47
HR	135.0	139.0	138.0	143.0	144.0	145.0	138.0	137.0
	7.8	8.0	6.4	8.1	7.3	8.8	7.4	7.8
R	0.99^{a}	1.00^{b}	0.94^{a}	0.96^{b}	1.06^{c}	1.04^{d}	0.97^{c}	0.98^{d}
	0.03	0.01	0.01	0.02	0.01	0.02	0.01	0.01

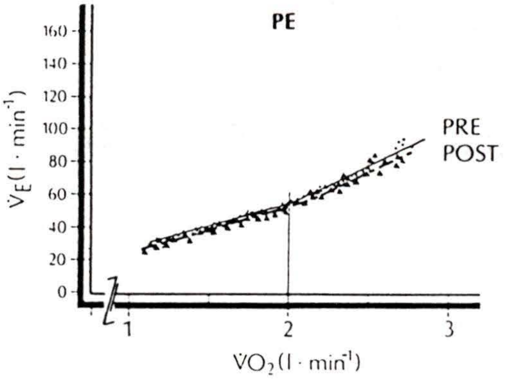
Values are means + SE

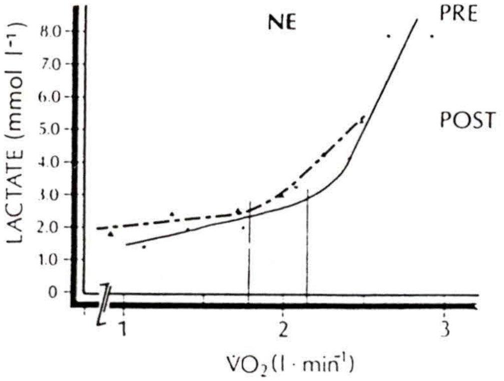
Paired letters indicate significant differences between tests ($p < 0.05$)

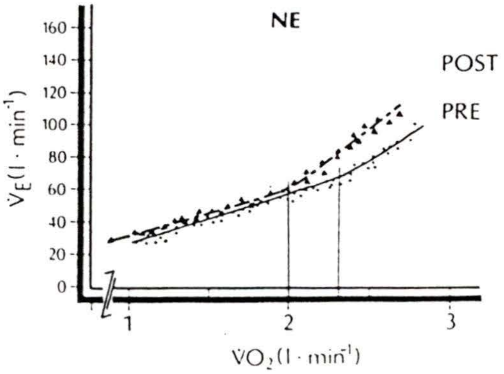
Figure 2 - Pre- and post- lactate and ventilation threholds for the prior exercised (PE) and non-exercised (NE) legs (subject 8)

Mean LA concentrations at LT and VT were different for both PE and NE conditions. Mean VO_2 values showed no significant differences at LT or VT.


PE
PRE
POST
LACTATE (mmol l⁻¹)
8.0
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0
1
2
3
$\dot{V}O_2$ (l · min⁻¹)


PE
PRE
POST
$\dot{V}_E$ (l · min⁻¹)
160
140
120
100
80
60
40
20
0
1
2
3
$\dot{V}O_2$ (l · min⁻¹)


NE
PRE
POST
LACTATE (mmol l⁻¹)
8.0
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0
1
2
3
$\dot{V}O_2$ (l · min⁻¹)


NE
POST
PRE
$\dot{V}_E$ (l · min⁻¹)
160
140
120
100
80
60
40
20
0
1
2
3
$\dot{V}O_2$ (l · min⁻¹)

SUMMARY

Venous lactate concentrations were significantly reduced ($p < 0.05$) at the LT and VT when comparing continuous versus discontinuous cycling, during glycogen reduction due to prior exercise, and when 1- and 2-legged work was performed.

A reduced lactate concentration at threshold had no effect on ventilation or oxygen consumption. VO_2 ($l\ min^{-1}$) was similar at LT and VT under the experimental conditions tested. This suggests that venous blood lactate is not the drive for V_E or VO_2 at threshold. It is further suggested that O_2 cost or power output in the working muscles may be related to these threshold points.

The lactate and ventilatory threshold do not demonstrate a cause and effect relationship.

Since lactate levels, and therefore LT, can be manipulated during prior exercise, an arbitrary lactate concentration of 4 mmol l^{-1} to represent threshold is not justifiable.

VO_2 ($l\ min^{-1}$) during 1-legged exercise was able to reach 74-85% of 2-legged VO_2 max while using only half the exercising muscle mass. This suggests that central (circulatory) factors are primarily responsible for limiting VO_2 max.

Glycogen levels during the prior exercise conditions demonstrated a 46.7% and 36.4% reduction in the prior exercised and non-exercised legs, respectively. This suggests that both central and peripheral (cardiovascular) factors are crucial in regulating the physiological responses to submaximal exercise.

Recommondations

1. Future research using the one-legged exercise model, to include additional invasive measurements (i.e. blood glucose, hormonal levels), would be valuable in providing a greater understanding on the mechanisms involved (central circulatory or peripherally within the muscle) during endurance performance.

2. It is recommended that addition research examine the relationship between the lactate and ventilatory threshold by observing oxygen cost and power output as the independent variables.

3. It is suggested that other prior exercise studies be performed to further substantiate the disuse of an arbitrary blood lactate (4 mmol l^{-1}) concentration.

APPENDIX A

UNIVERSITY OF VICTORIA
P.O. BOX 1700, VICTORIA, BRITISH COLUMBIA, CANADA V8W 2Y2
TELEPHONE (604) 721-7211, TELEX 049-7222
Sport and Fitness Testing Centre

School of Physical Education
721-8373

INFORMED CONSENT
FOR
PHYSIOLOGICAL ASSESSMENTS

In order to assess physiological function(s) the following laboratory assessments will be performed:

Lab Initial	Subject Initial	
___	___	Submaximal Cardio-Respiratory Function

You will exercise on an ergometer at 75% of predicted maximum heart rate. The following indicated variables will be measured:

a) ventilatory responses ___ c) thermoregulatory responses ___

b) heart rate responses ___ d) other ____________

___	___	Maximal Cardio-Respiratory Function

You will exercise on an ergometer with progressively increasing loads to elicit maximal responses in the following indicated variables:

a) oxygen consumption ___ c) ventilation ___

b) heart rate ___ d) other ____________

___	___	Submaximal and/or Maximal Muscular Contractions

You will perform submaximal or maximal muscular contractions in the following modes:

___isometric ___isotonic ___isokinetic ___eccentric

___	___	Blood Chemistry

Blood samples may be taken prior to, during, or post-exercise by:

a) venipuncture ___ b) finger tip prick ___

Lab Initial	Subject Initial	
___	___	Body Composition Lean body mass and percent body fat may be assessed by: a) anthropometric measures ___ b) body densiometry ___
___	___	Tests will be administered by qualified personnel under the direct supervision of the investigator(s).
___	___	Blood samples will be taken by a qualified laboratory technician or registered nurse.
___	___	Training will be monitored by the investigator(s) or trained assistants.
___	___	Test and training data and results will be treated in a confidential manner and used only to describe group responses.
___	___	Absolute confidentiality of individual results will be maintained unless specific approval has been given to other use of the material by each subject, or guardian where necessary.
___	___	While it is highly unlikely that a subject should be injured or taken ill during a test or training session, lab personnel are trained in emergency procedures and emergency equipment is on-site at all times.
___	___	All laboratory activity will be completed proximal to medical and/or paramedical assistance.
___	___	The maximal exercise loads imposed will not exceed those which might be expected of an athlete during sports performance.

I have read the above and agree to participate in this research project/ fitness appraisal at my own risk. I regularly take part in strenuous physical activity at least as intense as these tests. I realize that I may expect a thorough explanation and/or demonstration of any procedures and that I may terminate participation at any time in any or all procedures of my own volition.

Having voluntarily assumed participation and the risks thereof, in the project, I hereby disclaim and release the University of Victoria, its agents, servants or employees, including all personnel involved in the research project fitness appraisal from any and all liability that might otherwise arise as a result of my participation as a research subject in this study/or fitness appraisal.

NAME: ______________________________ DATE: ______________
(please print)

SIGNATURE: ______________________________

I, the undersigned guardian, am guardian of ______________________, the intended subject. I have discussed the experimentation with the subject and have read the material supplied by the experimentors. I agree on behalf of the subject to permit his/her participation on the terms and subject to the waiver and release of the University of Victoria hereinbefore set out.

GUARDIAN'S SIGNATURE: ______________________________
(where applicable)

DL:gs
84/10/25

APPENDIX B

ALLOWABLE FOOD LIST

(low carbohyrate diet)

The following is a list of 'allowable foods' that can be consumed during meals. Please select those food you wish to eat, but only in moderation. The quantity listed beside each food item is the amount permitted per meal.

Bacon - 3 strips
Cheese - cream, swiss, American
Eggs - 2
Ham - canned (4-6 oz.)
Heart - beef braised
Kidney, beef, lamb (4-6 oz.)
Lettuce
Lobster (3-4 oz.)
Margarine or butter (2 tbsp.)
Olives
Onions - green and raw, mature and dry
Parsley
Green peppers - raw
Mayonnaise (1 tbsp.)
Sauces - hollandaise, tartar
       - low calorie salad dressing (1 tbsp.)
Sausage - frankfurter, liverwurst, pork, salami, vienna
Luncheon meat - pork (cured, canned, packaged)
Shrimp (2-3 oz.)
Tongue
Dietary drinks (less than 1 cal./oz.)
Whole milk (1 cup)
Yogurt - plain (1 cup)
Chicken (2-3 pieces)
Bread - 2 slices
Tuna/Salmon (3 oz.)

APPENDIX C

REVIEW OF LITERATURE

It has been suggested that the ability of an individual to perform endurance activity depends largely on his capacity to consume, transport and utilize oxygen in the working muscles (Rusko et al., 1978). Maximal aerobic power, or maximal oxygen consumption (VO_2 max) has been used most extensively as a criterion for cardiovascular fitness and performance capacity for endurance activities (Costill, 1967; Saltin & Astrand, 1967; Astrand & Rodahl, 1977). A number of researchers have stated that VO_2 max is the single best predictor of endurance performance (Costill, 1967; Burke et al., 1977). Rusko et al.(1978) suggested that VO_2 max might be the most important determinant of performance when large muscles are used during short and long periods of time (i.e. a high correlation, r=0.94, has been found between VO_2 max and running performance over a wide range of distances) (Costill et al., 1973; Karlsson, 1971).

There is still considerable controversy however, as to what factors determine the maximal aerobic power of an individual. While it seems likely that the primary factor responsible for increases in VO_2 max is an improved cardiovascular system, it appears unlikely that increased O_2 delivery improves endurance performance (Holloszy & Coyle, 1984).

Empirical evidence suggests that metabolic changes at cellular level accompany (or cause?) an increased VO_2 max and endurance capacity. These changes include:

1. an increase in the size and number of the mitochondria resulting from endurance training (Hickson et al., 1977; Holloszy, 1973; Gollnick et al., 1971). With an increase in mitochondrial size and number, oxidation of pyruvate and free fatty acids are enhanced.
2. a greater extraction of O_2 from the blood at high intensity work rates, indicating a greater utilization of O_2 by the exercising tissues as a result of alterations in mitochondrial composition (Fox et al., 1975; Karlsson et al., 1967).
3. enhanced oxidative capacity of both fast and slow twitch muscle fibers (Gollnick et al.,1973).
4. lower lactate production during submaximal exercise due to adaptive increases in mitochondrial enzymes (Holloszy & Coyle, 1984)

Many training models have been employed to determine whether peripheral or central factors limit VO_2 max. Karlsson et al. (1975), and Saltin et al. (1976), using a one-legged model suggested that central cardiovascular improvements are not elicited unless peripheral factors are also improved. They concluded that a very close 'interplay' exits between peripheral and central factors. Their data illustrates that improvements in VO_2 max occurred in both one- and two-legged exercise. Although the two-legged exercise produced slightly greater increases in VO_2 max, the ratio between the two was small (both pre- and post- training). Gleser (1973) however, concluded that peripheral factors limited VO_2 max. He argued that neither before nor after one-legged training did cardiac output reach maximum in one- versus two-legs. Davies and Sargeant (1975) also suggested that one-legged work is limited primarly by the periphery, but in two-legged exercise the limitation in VO_2 max is dependent on the capacity of the central cardiovascular system to transport oxygen to the effective muscle mass. Likewise, it has been suggested that any factor impairing the metabolic processes at the cellular level can limit an individual from performing prolonged exercise (Wenger & Reed, 1976). More research is required before a definite answer to this question is available.

Research reveals that VO_2 max can be elevated through a regularly maintained training program (Williams et al., 1967; Wenger & MacNab, 1975; Davis et al., 1976; Hickson et al.,1977; Ekblom, 1969; Smith & Wenger, 1981). The magnitude of VO_2 max increase is related to four primary factors: initial fitness level, and intensity, duration, and frequency of training (Fox et al., 1975; Hickson et al., 1977; Rowell, 1974; Atomi & Miyashita, 1976; Ekblom, 1969). The mode of training has also been suggested as a factor for elevating aerobic power (Smith & Wenger, 1981). Of the primary factors intensity of training is suggested as the key independent variable which determines changes in the training effect (Burke, 1975; Davies & Knibbs, 1971; Crews & Roberts, 1976; Katch et al., 1973). These factors, in combination with progressive overload (Fox et al., 1975; Smith & Wenger, 1981, Davies & Knibbs, 1971), are required to elicit improvements in fitness. Endurance training can be either interval or continuous in nature.

It has been documented (Davies and Knibbs, 1971 Hickson et al., 1977; Saltin & Astrand, 1967) that VO_2 max is genetically limited and therefore cannot be increased substantially with further training. However, research indicates that improvements in other physiological variables result with additional endurance training (Pollock, 1973; Ekblom, 969; Saltin & Astrand, 1977). Costill et al. (1973) stated that VO_2 max alone does not adequately predict a winning performance, and that other components of fitness must therefore be related.

It appears the most significant physiological gain is in the athlete's ability to use a higher percentage of his maximal aerobic power (Sjodin et al., 1982) without the accumulation of lactate in muscle and blood. Wasserman et al. (1973) proposed using the term 'anaerobic threshold' (AT) to describe the metabolic events associated with the transition from aerobic to anaerobic processes. They defined AT as the 'level of work or rate of oxygen consumption just below that at which metabolic acidosis and its associated gas exchanges occur'. These researchers suggest that the initial increases in lactate coincide with a non-linear increase in minute ventilation (V_E), an increase in end-tidal O_2 tension ($PETO_2$) without an increase in end-tidal CO_2 ($PETCO_2$) tension, and an increase in CO_2 production. There also appears to be a linear relationship between plasma lactate concentration and CO_2 output (Taylor and Jones, 1979). This relationship exists because lactate accumulation in plasma is accompanied by hydrogen ion (H^+) efflux from muscle. Hydrogen ion efflux increases CO_2 production which stimulates an increase in V_E (Whipp, 1978). It is this relationship which allows the ventilation threshold to be used as a indication of the lactate threshold (Jones & Ehrsam, 1982).

Although the original work of Wasserman et al. (1973) has helped to expand our knowledge and understanding of the disturbances in gas exchange associated with exercise metabolic acidosis, other researchers have demonstrated that AT, as defined by Wasserman, is an inadequate threshold level for testing or training endurance athletes (Davies & Knibbs, 1971; Kindermann et al., 1979; Skinner & McLellan, 1980). Therefore, the presence of two 'breakaway' points from linearity in the relationship between V_E and VO_2 has been used to define AT (Skinner and McLellan, 1980; Kindermann et al., 979). Thus, the selection of an appropriate definition is one focus of attention. Kindermann et al. (1979), in agreement with other researchers (Costill, 1970; Costill et al., 1796; Rusko et al., 1980) have shown that highly trained endurance athletes can work at high intensities (80-85% VO_2 max) for 45-60 minutes even in the presence of elevated lactate levels. Therefore, the work intensity required to reach such a level of 'metabolic acidosis' as described by Wasserman and co-workers (1973) would be equivalent to performing physical activity similar to that for rehabilitation purposes, or for the prognosis of cardiorespiratory and pulmonary disease patients. MacDougall (1977) defines AT as the 'workload intensity at which blood levels of lactic acid begin to rise significantly above resting values during steady state exercise'. This definition seems more reasonable because athletes have demonstrated the ability to run at high intensities for prolonged periods on a treadmill (70-85% VO_2 max).

Research performed in the Scandinavian countries have used a different terminology to describe this metabolic event. The Onset of Blood Lactate Accumulation (OBLA) is used to indicate the threshold level (60-70% VO_2 max) where anaerobic processes increase (Sjodin et al., 1982). This threshold level is similar to that defined by Hughes et al. (1982) (who use the terminology lactate and ventilation thresholds), MacDougall (1977), Kindermann et al. (1979), and Skinner and McLellan (1980). In agreement with Thoden et al. (1983), Sjodin and collaborators (1982) use only muscle and blood lactate to represent AT, and question whether the changes in respiratory variables are a valid criterion measure for determination of the 'lactate breaking point' and/or the anaerobic threshold as suggested by Wasserman et al. (1973). Sjodin and co-workers believe it is difficult to use respiratory measurements to determine AT because the cause of lactate appearance in blood is still obscure. Originally, it was thought that lactate production was due to insufficient O_2 availability, illustrating tissue hypoxia. Recent studies have demonstrated that lactate is present when sufficient levels of NAD^+ are present, indicating that O_2 is available for ATP production (Nagle, 1973; Skinner & McLellan, 1980). Therefore, lactate is present in the presence of O_2, suggesting it seems unlikely that a hypoxic condition exists (Holloszy & Coyle, 1984).

Hughes et al. (1982) have also suggested that the 'concept of a ventilatory anaerobic threshold should be discarded because of the absence of any direct data relating muscle anaerobiosis during exercise to the ventilation AT'. Studies by Green et al. (1983) and Simons et al. (1983) have also demonstrated that the ventilation anaerobic threshold (VAT), as determined by the relationship between V_E and VO_2, are not coincidental with the lactate threshold.

In contradiction to the above authors, Davis et al. (1976) have shown that routine laboratory measures of gas exchange in three modes of exercise (arm cranking, leg cycling, treadmill walk-running), using reliable rapidly responding O_2 analyzers, have found test re-test correlations of r=0.95 between gas exchange parameters and blood lactates for the determination of AT. The correlation utilizing minute ventilation alone was found to be r=0.88. They concluded that gas exchange AT is a valid and reliable indirect method for detecting the onset of lactate acidosis during incremental exercise.

Recently, others have criticized AT as an invalid measure for detecting anaerobic metabolism because of the numerous criteria used to define this event. Yeh et al. (1983) in a review of the literature revealed these criteria to include: an increase in both arterial and venous lactate levels; a decrease in bicarbonate levels; an arbitrary lactate value; and noninvasive measures. Their research showed that significant differences existed between arterial and venous lactate concentrations, and further that no threshold phenomenon were detected.

Others however, have used AT as a criterion measure for submaximal fitness and propose that it is an accurate predictor for endurance performance. Sjodin et al. (1982), Weltman et al. (1978), LaFontaine et al. (1981), and Davis et al. (1979) have shown that OBLA and AT correlate significantly (r=0.90-0.99) with the performance capacity of trained athletes during distance events. Some suggest it may be a more accurate predictor of endurance than VO_2 max (Sjodin et al., 1982).

A number of studies have also illustrated that the anaerobic threshold can be increased through training. Increases in AT of 4-44% have been recorded following 4-16 weeks of training (Williams et al., 1967;; Ekblom et al., 1968; Ekblom, 1969; Davis et al., 1976).

Glycogen depletion studies by Gollnick et al. (1973), Edgerton et al. (1973) and Essen (1978) revealed the fundamental characteristics of different muscle fiber types and how their recruitment patterns during exercise at different intensities affect AT. Slow twitch muscle fibers (ST) are highly oxidative fibers and contain a greater amount of heart-specific lactate dehydrogenase (LDH) isozyme (H-LDH) which favors the oxidation of lactate to pyruvate. Fast twitch muscle fibers (FT) are highly glycolytic fibers and contain a greater amount of muscle specific LDH isozyme (M-LDH) which favors the reduction of pyruvate to lactate (Skinner & McLellan, 1980). Thus, lactate production and determination of AT is partially dependent on which muscle fibers are recruited. During the early stages of exercise little or no lactate is produced, indicating the recruitment of predominantly ST muscle fibers. As exercise intensity increases, more FT are recruited, causing an imbalance between pyruvate production and oxidation. This results in an increase of lactate above resting levels (2 mmol l^{-1}), which is in agreement with findings by Sjodin (1976) who demonstrated that lactate formation may be related to the metabolic profile of the muscles. Likewise, Ivy et al. (1980), and recently Gleim et al. (1984), suggest that fiber composition of the exercising muscles is related to the level of VO_2 at which AT occurs. Athletes with a greater percentage of ST type I fibers have a higher AT-%VO_2 max level than those with more FT type II fibers. This evidence suggests that there may be a genetic and cellular (Smith, 1981) basis to the anaerobic threshold.

The effects of glycogen depletion (GD) have also helped to expand our knowledge on substrate utilization in exercising muscle. Depleted glycogen stores induced by exercise and diet increase free fatty acid (FFA) oxidation, part of which is regulated by increasing lipoprotein lipase (LPL) activity (Jacobs, 1981). Jacobs demonstrated that LPL activity increases as muscle glycogen concentration decreased following a fat-protein diet and exercise regimen. Peak lactate levels become reduced as a result of fatty acid oxidation, which in turn elevates muscle citrate concentration. Increased citrate levels inhibit phosphofructokinase (PFK) in glycolysis and therefore plays an important role in substrate utilization (Essen, 1977). In addition to metabolic alterations with glycogen depletion through diet and exercise, hormonal acitivity is markedly changed. The plasma concentration of glucagon, growth hormones, epinephrine and plasma catecholamine levels increase and impede glycogen replenishment (Galbo et al., 1977).

The effects of reduced muscle glycogen content on the AT have been reported (Hughes et al., 1982). It was shown that lactate levels, V_E, R, and glucose levels were altered by glycogen depletion (GD), which in turn has altered the lactate and ventilation thresholds. Likewise, work by Heigenhauser et al. (1983) found significant increases in V_E, VCO_2, VO_2, HR and reduced lactate concentration. Although HR was increased, cardiac output displayed no change between the GD and control conditions, suggesting a 'neurogenic' drive is possibly responsible for HR and V_E increases. Similarly, recent work by Gleim et al. (1984) support that other factors may elicit such metabolic changes at threshold. They showed that AT may represent a pivotal point during progressive exercise due to plasma osmolality and the hormonal changes described above. Thus, a combination of neurogenic, hormonal, and hemodynamic events may be occurring simultaneously.

From the review of literature, maximal aerobic power has demonstrated that it is a valid and reliable endurance parameter. The anaerobic threshold however, has been implied as an aerobic parameter useful for predicting performance, designing training programs, and as a metabolic link between aerobic-anaerobic transition of energy production. It is hoped that this research has provided a greater understanding of the factors affecting the measurement of the anaerobic threshold.

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